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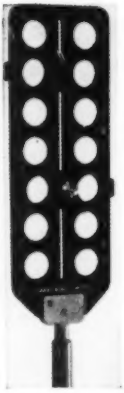

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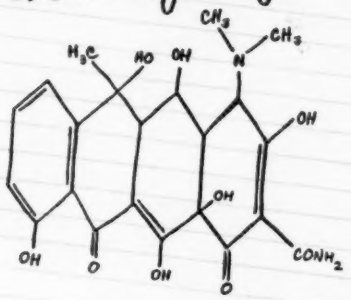

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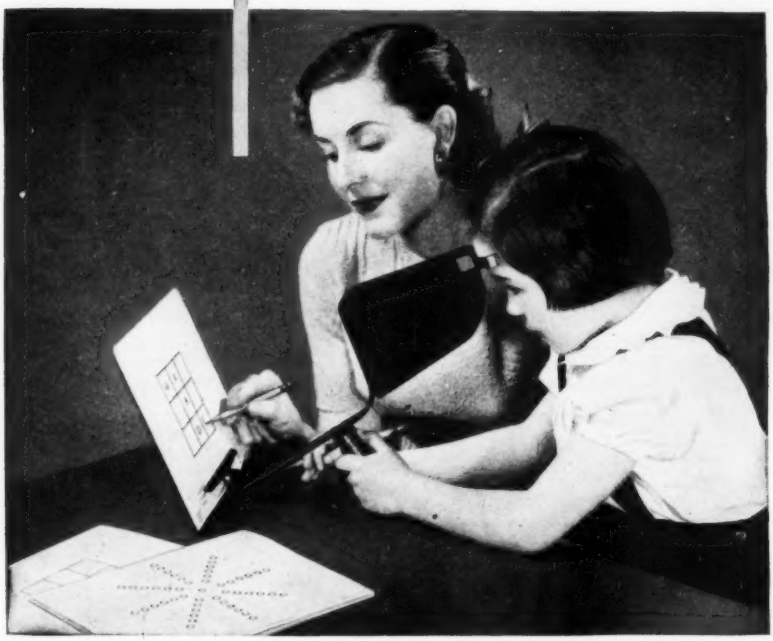

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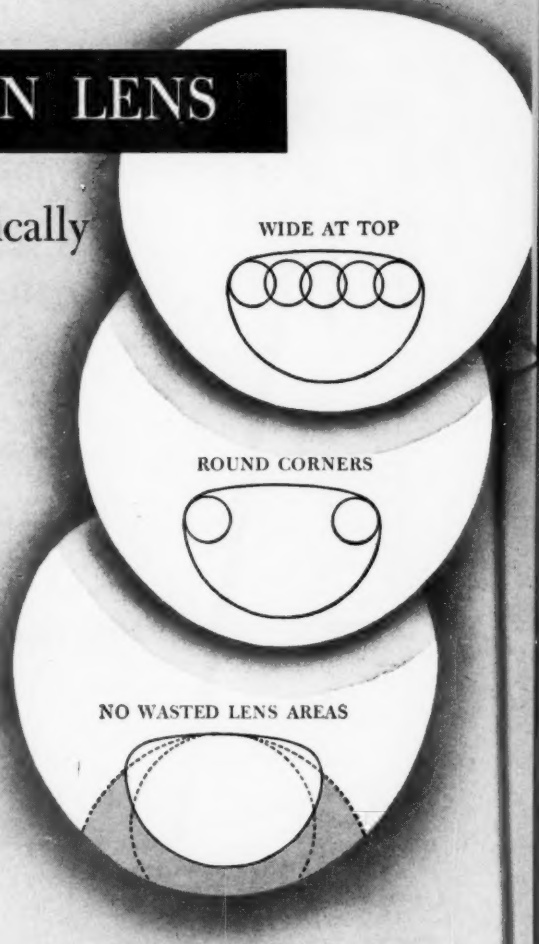

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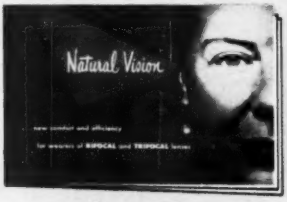

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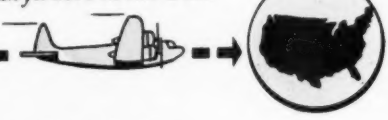

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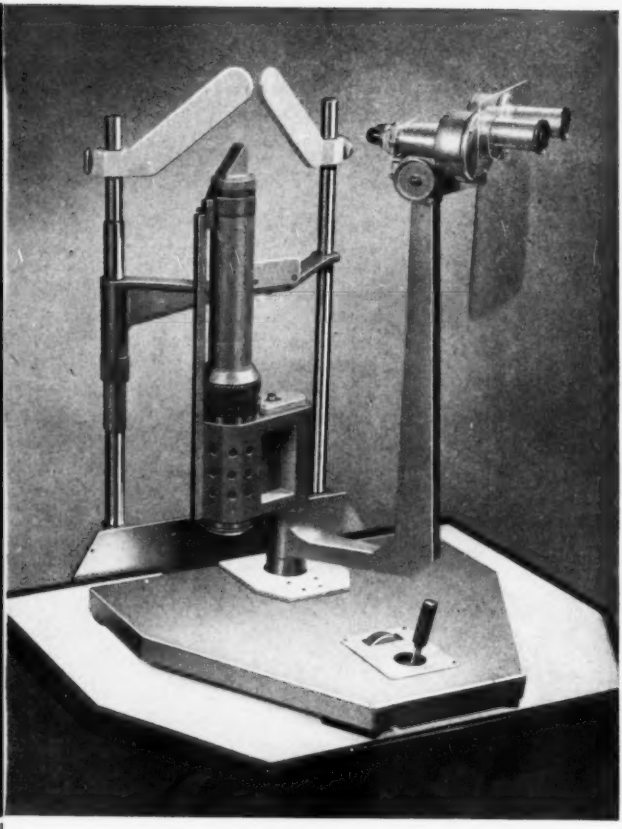

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1. Scheie, H. G., Tyner, G. S., Buesseler, J. A., and Alfano, J. E., *J. A. M. A. Arch. Ophth.* 45:301, March 1951.

2. Leopold, I. H., Purnell, J. E., Cannon, E. J., Steinmetz, C. G., and McDonald, P. R., *Am. J. Ophth.* 34:361, March 1951.

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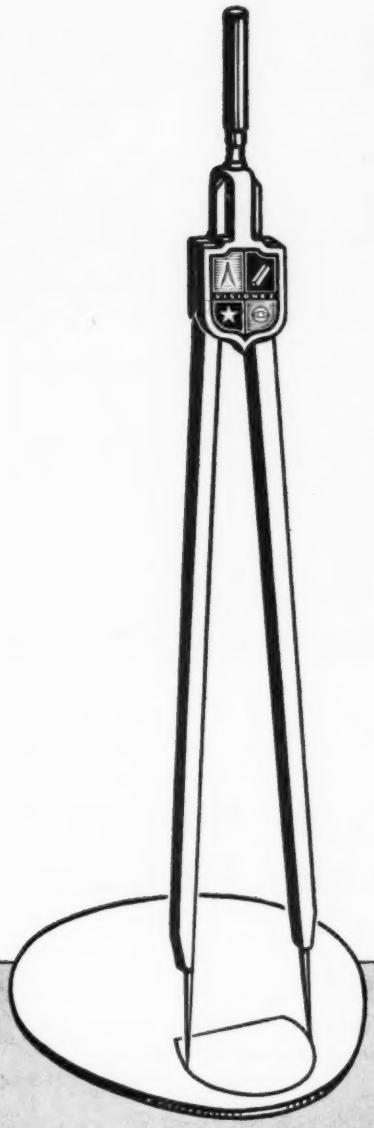

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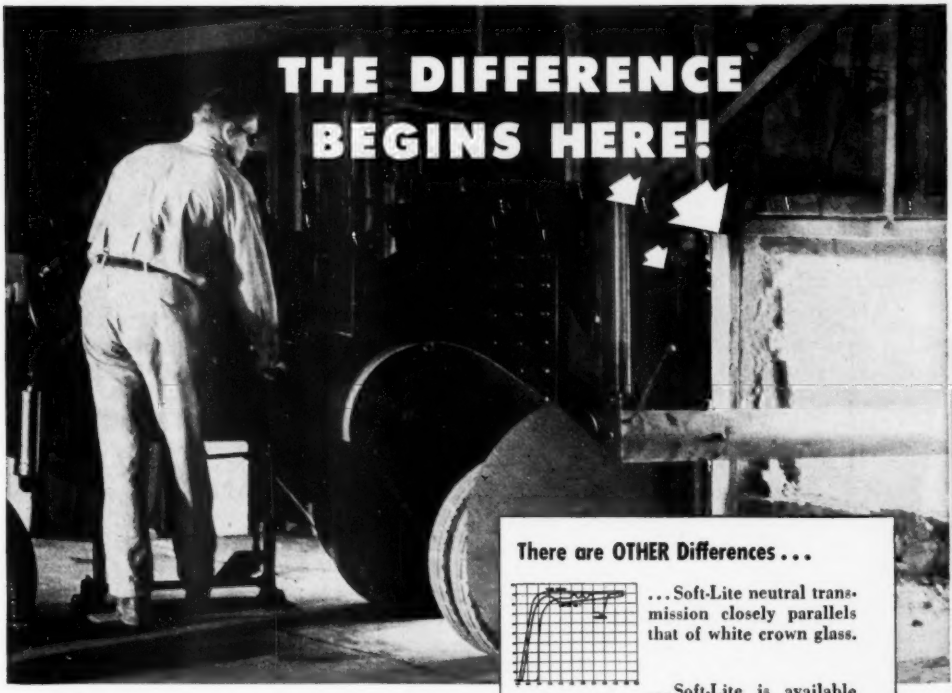

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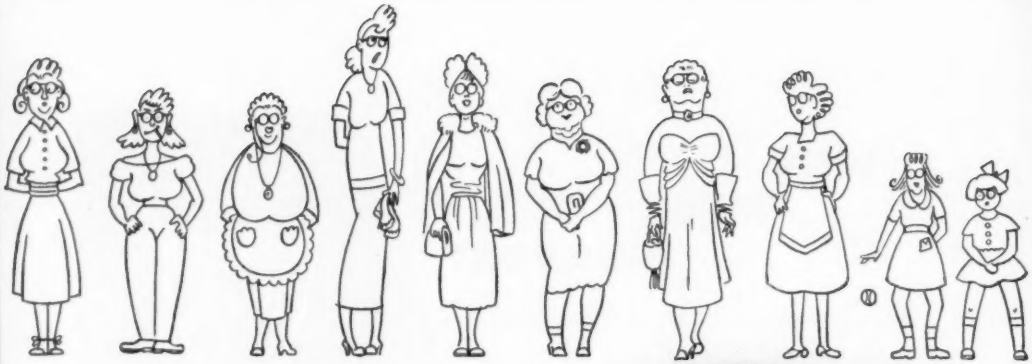

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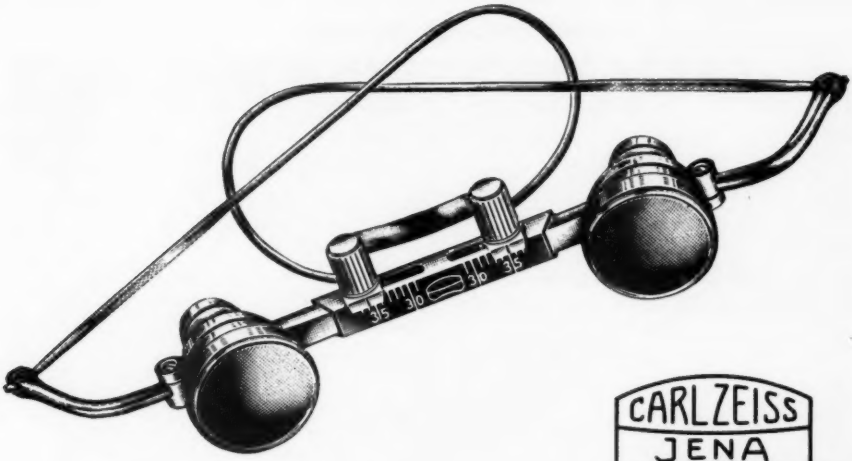

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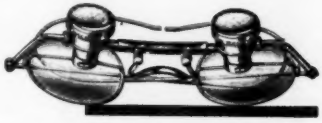

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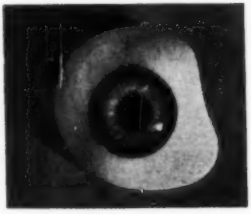

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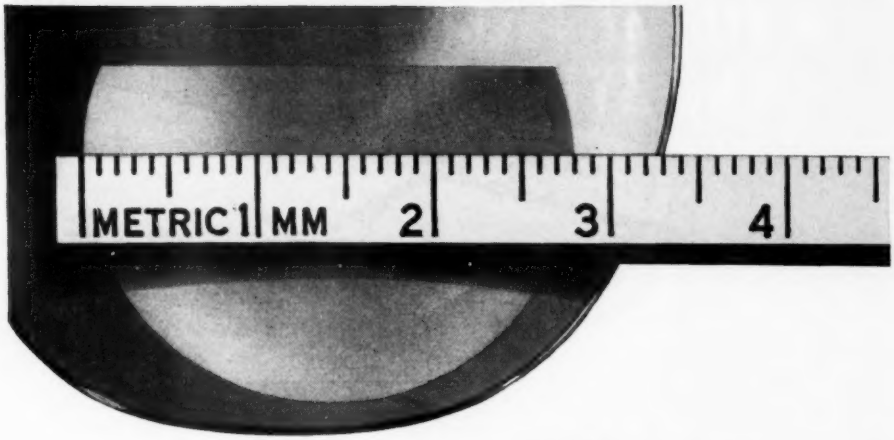

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THE DIABETIC: HIS VISUAL PROGNOSIS

The Third John E. Weeks Lecture

FREDERICK C. CORDES, M.D.
SAN FRANCISCO

IT IS A great honor to have been asked to deliver the third John E. Weeks Lecture.

John E. Weeks was one of the small group of illustrious ophthalmologists who laid the foundation for the present high standard of our specialty in America. His contributions are so many that it would hardly be possible to enumerate them here. I shall mention only one: the epoch-making identification in 1886 of the organism which bears his name, the Koch-Weeks bacillus. The microscope he used in this work today occupies a place of honor in the living room of his nephew, the Los Angeles ophthalmologist, Dr. Carrol Weeks.

My first contact with Dr. Weeks was in St. Louis in 1922, when he examined me in pathology for the American Board of Ophthalmology. He appeared cold and severe, and I was awed by him. In later years, after his retirement, it was my privilege, during short winter vacations in Palm Springs, to sit with him after dinner in the lobby of the Desert Inn. Here I heard the exciting stories of his early hardships and struggles and of the early days of American ophthalmology—stories that should be a source of inspiration to us and to the ophthalmologists of the future. Here, too, I learned that under the somewhat severe exterior was a warm heart in a modest, retiring man, whose emotions were deeply stirred by the tributes he received from his colleagues.

I cannot let pass this opportunity to pay my respects to Mrs. Weeks. Her implicit faith in her husband's ability gave him the confidence he needed during his early years, and I have it on the best authority that she was also his favorite fishing companion. May I add that her charm and graciousness have endeared her to all who have known her?

Dr. Weeks's fame was world-wide before his retirement, but it was his last 25 years in Portland that left an indelible impression on the ophthalmology of the Pacific Coast. Dr. Knapp said of him that he lived long but never grew old. It was this retained ardor and keenness of mind that prompted his efforts on behalf of western ophthalmology. Although we have all been benefited, it is doubtless here at the University of Oregon that his influence has left its most permanent imprint. The funds he gave for the building of an adequate library is but one example of the quality of his interest and generosity.

The art work was made possible by the E. S. Heller Fund.

From the Department of Ophthalmology of the University of California School of Medicine at San Francisco.

The presence of this great man, his sage advice, and the lively interest he retained in both research and clinical ophthalmology must have been of inestimable value to Dr. Swan in building up the department of ophthalmology of the University of Oregon to its present very high level. As another of the men of the West Coast who was fortunate enough to come under this stimulating influence, I feel it a very great honor to have been given the privilege of presenting this, the third, John E. Weeks Lecture. May the presentation be worthy of the honor.

DIABETES: GENERAL CONSIDERATIONS

Insulin therapy has prolonged the expectation of life in the diabetic. One of the important consequences is that there are now more diabetics with the degenerative retinal changes characteristic of diabetes of long standing. This is borne out by Wagener's statistical studies. In 1921 he and Wilder [1] found retinopathy in approximately 8% of the diabetics at the Mayo Clinic; in 1945 [2] he found it in 29.6%. In persons who had had the disease less than 15 years the incidence was less than 11%; in those affected for more than 20 years it was 74%. Friedenwald [3] states that diabetic retinopathy now occurs three times as frequently in his private practice as it did in 1924, although the incidence of many ocular diseases has not changed in that time.

This dramatic increase in the number of patients with retinopathy prompted the present attempt to determine the visual prognosis of the diabetic. The first step was to review the current concepts of diabetic disease and its ocular manifestations.

Etiology.—Our present knowledge of diabetes mellitus is ably summarized by Doggart.[4] By definition, it is a metabolic disease in which there are symptoms of polyuria, polydipsia, voracious appetite, progressive loss of flesh and strength, and a tendency to fatal termination. It is due essentially to an imbalance between insulin and its antagonists, the hormones produced by the pituitary gland, the thyroid gland, and the adrenal cortex. As a rule there is absolute, as well as relative, deficiency of insulin, but the relative deficiency is the decisive factor.

In 1930 Houssay and Biasotti [5] found that the hypersensitivity to insulin created by the experimental removal of the pituitary gland could be abolished by the injection of anterior pituitary extracts. They found, further, that dogs deprived of both the pancreas and the pituitary lived longer than dogs from which only the pancreas had been removed. Injection of anterior pituitary extract into the intact animals led to glycosuria because of the antagonism of the pituitary extract for insulin. Haist and his group [6] found that the glycosuria could be prevented if sufficient quantities of insulin were given simultaneously. In 1945 Price and his co-workers [7]

1. Wagener, H. P., and Wilder, R. M.: The Retinitis of Diabetes Mellitus, J. A. M. A. **76**:515-517 (Feb. 19) 1921.

2. Wagener, H. P.: Retinopathy in Diabetes Mellitus, Prof. Am. Diabetes A. **5**:201-216, 1945.

3. Friedenwald, J. S.: Diabetic Retinopathy, Am. J. Ophth. **33**:1187-1199 (Aug.) 1950.

4. Doggart, J. H.: Retinopathy in the Diabetes, Tr. Ophth. Soc. Australia **9**:139-153, 1949.

5. Houssay, B. A., and Biasotti, A.: Les troubles diabétiques chez les chiens privés d'hypophyse et de pancréas, Compt. rend. Soc. biol. **105**:124-126 (Oct. 16) 1930.

6. Haist, R. E.; Campbell, J., and Best, C. H.: Prevention of Diabetes, New England J. Med. **223**:607-615 (Oct. 17) 1940.

7. Price, W. H.; Cori, C. F., and Colowick, S. P.: Effect of Anterior Pituitary Extract and of Insulin on Hexokinase Reaction, J. Biol. Chem. **60**:633-634 (Oct.) 1945.

demonstrated that both insulin and anterior pituitary hormone are concerned with the preliminary phosphorylation of glucose, which is a prerequisite to the utilization of the sugar.

Types.—There are two recognized types of diabetes: the hyperfunctional type, in which the pancreas is unable to deal with a hyperproduction of sugar, caused by hypophyseal stimulation, and the rarer, hypofunctional type, which is primarily a pancreatic disease. Appel [8] states that 70% of diabetics have the hyperfunctional type; of this group, 80% are hypertensive. In patients with the hypofunctional type hypertension occurs no more frequently than it does in the same age group of non-diabetic patients.

Incidence.—Diabetes occurs more frequently in women than in men and develops characteristically in two distinct age groups: in a small group, of infants, children, and adolescents; and in a much larger group, of adults, usually between the ages of 40 and 70. Heredity is a definite factor, and the tendency to diabetes is probably transmitted as a simple Mendelian recessive. There seems to be a racial difference in susceptibility, the French, for example, being less susceptible than people of other countries. These differences may well be attributable less to race than to the dietary habits of the people, however; some racial or national groups tend to overeat habitually. Certainly, obesity is a predisposing factor.

Symptoms.—The two earliest symptoms of diabetes are polyuria and polydipsia; the fluid mobilized from the tissues in order to dilute the sugar of the blood is replaced by the additional fluid intake. The next symptom is usually loss of strength, often associated with shortness of breath. The patient has an unusual appetite, and frequently there is gradual loss of weight. Pains in the wrists and tingling of the fingers, as a result of peripheral neuritis, are at times among the early symptoms. There is often long-continued emotional stress, probably related to the fact that diabetes is frequently associated with hyperthyroidism. Two specific manifestations are Kimmelstiel-Wilson disease (glomerulosclerosis)[9] and retinopathy.

Metabolic Sequence.—The metabolic sequence, as outlined by Doggart,[4] is as follows:

Imbalance of the endocrine control of the carbohydrate metabolism signifies relative insulin deficiency. This is followed by alteration of the liver threshold value for adjustment of the blood glucose content, until the glucose overtops the renal dam and excess sugar escapes into the urine. Hyperglycemia spurs the islets of Langerhans into excessive activity, which accelerates their destruction and establishes a vicious circle. Polyuria is the natural result of the extra water taken in to aid the transport of the sugar load along the blood vessels. Owing to the relative insulin deficiency, the tissues are incapable of utilizing the sugar with which they are flooded; yet the production of sugar persists to provide the sole readily available source of energy for the tissues, especially the cells of the central nervous system. Catabolism of proteins now takes place to replace the wasted sugar. An increase in the nitrogenous constituents of the urine, together with loss of weight and a markedly increased appetite, are the sequels. Ultimately, fat metabolism is deranged, and acetoacetic acid and allied substances upset the mineral balance of the blood and

8. Appel, W.: Hochdruck und Diabetes, Klin. Monatsbl. Augenh. **116**:225-237, 1950.

9. Kimmelstiel, P., and Wilson, C.: Intercapillary Lesions in the Glomeruli of the Kidney, Am. J. Path. **12**:83-98 (Jan.) 1936.

emerge in the urine and in the expired air. When the alkaline bases in the plasma become incapable of carrying normal amounts of carbon dioxide from the tissues to the lungs, the patient becomes air-hungry, lapses into coma, and dies unless relieved artificially.

Treatment.—Soon after its isolation by Maclead, Banting, and Best, in 1922, insulin became available for clinical use, and physicians learned how to regulate the dosage for various types of patients. With proper diet, control of weight, insulin therapy as indicated, and the administration of vitamin B complex (Biskind and Schreier [10]), it is now possible to control diabetes in most cases, and the inconvenience of too frequent injections can be avoided by combining the insulin with protamine and zinc chloride, which spread its action over relatively long periods.

Prognosis.—The work of Robbins and Tucker [11] indicates that at present diabetes does not diminish the average expectation of life. Life insurance statistics [4] emphasize the menace of the related obesity, however; and there are several serious complications to be reckoned with. In a study of 221 diabetic patients who had survived at least 15 years, Martensson [12] found arterial hypertension in 57%, coronary arteriosclerosis in 43%, and renal disease in 34%; in a study of 644 patients, Engel [13] found that 25% of those under 40 years of age had renal disease, and White and Waskow [14] found evidence of nephritis in 50% of patients who had survived 20 years of diabetes acquired in childhood. One the other hand, when the diagnosis of diabetes induces the patient to control his weight and to take better care of himself, the disease may actually be a factor in the prolongation of life. Thus, only if contracted in childhood or adolescence should diabetes now be said to "load the scales against longevity."

OCULAR COMPLICATIONS

The ocular manifestations of diabetes have been well defined and will be described briefly before the discussion of visual prognosis is undertaken.

Wrinkling of the Posterior Cornea.—In both normal and diabetic persons there may be wrinkling of the posterior cornea, but according to Waite and Beetham [15] it occurs in 26% of diabetics and in only 10.5% of nondiabetics. The wrinkling is visible only with the slit lamp and is at the depth of Descemet's membrane.

Weakness of Accommodation.—Approximately 20% of diabetics show weakness of accommodation.[15] According to Duke-Elder,[16] the insufficiency may amount to as much as 3 or 4 D. and is occasionally accompanied by complete paralysis. It occurs oftenest in young people, and the onset is usually sudden. The weakness is

10. Biskind, M. S., and Schreier, H.: On the Significance of Nutritional Deficiency in Diabetes, J. Exper. Med. & Surg. **3**:299-316 (Nov.) 1945.

11. Robbins, S. L., and Tucker, A. W., Jr.: Cause of Death in Diabetes: Report of 307 Autopsied Cases, New England J. Med. **231**:865-868 (Dec. 28) 1944.

12. Martensson, J.: Prognosis of Diabetes Mellitus: Study of 221 Patients Surviving at Least 15 Years, Acta med. scandinav. **137**:335-354 (July) 1950.

13. Engel, A.: Sena diabetes komplikationer, Nord. med. **43**:902-908 (June) 1950.

14. White, P., and Waskow, E.: Clinical Pathology of Diabetes in Young Patients, South. M. J. **41**:561-567 (June) 1948.

15. Waite, J. H., and Beetham, W. P.: Visual Mechanism in Diabetes Mellitus: Comparative Study of 2002 Diabetics and 457 Non-Diabetics for Control, New England J. Med. **212**:367-379 (Feb. 28) 1935; 429-443 (March 7) 1935.

16. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 4, St. Louis, C. V. Mosby Company, 1949, p. 4437.

bilateral, but there is normal pupillary reaction. Rea [17] suggests that excessive glycogen deposits in the pigment epithelium of the ciliary bodies may interfere with normal function, or that there may be an altered condition of the lenses. The insufficiency tends to disappear when the diabetes is controlled.

Changes in Refraction.—It was Horner [18] who in 1873 first called attention to the sudden changes in refraction that occur in diabetes. His observation has since been confirmed many times. Duke-Elder [19] states that characteristically the changes come on suddenly and bilaterally; that myopia is associated with a rise, and hyperopia with a fall, in the sugar concentration of the blood, and that the hypermetropic change seems not to occur as an initial phenomenon, but to follow a myopic change. As the blood sugar concentration varies, the refractive state of the eye may change from hypermetropia to myopia and vice versa. The alteration in the refraction is frequently associated with astigmatic changes of from 2 to 3 D. Duke-Elder observed one case with —6 D. of astigmatism in one meridian and +9 D. in the other. There is evidence that in the terminal coma a much higher degree of myopia may develop. Bellows [20] observed that, in contrast to the rapid development of the hypermetropia, the regression to a normal state was gradual, generally requiring from two to four weeks. This observation has been confirmed by Granström,[21] Waite and Beetham,[15] O'Brien and Allen,[22] and others.

There have been a number of theories as to the cause of these refractive changes. Elschnig's report [23] of a case of unilateral aphakia in which refractive changes developed only in the phakic eye limits our consideration to the lens. Duke-Elder [19] attributes transitory myopia to the entering of fluid into the lens osmotically, as a result of a variation in the sugar concentration of the blood and tissue fluids. When the fluid enters the lens the nucleus fails to take it up. This creates a difference in refractive index between the lens cortex and its nucleus and contributes further to the refractive change. When the diabetes is treated the circumstances are reversed: There is drainage of fluid from the lens, and the patient becomes relatively hypermetropic. Granström,[21] on the other hand, believes that hydration of the nucleus is responsible for the hypermetropic change. According to his observations, and to those of Lawrence, Oakley, and Barne,[24] and others, reversible changes in the lens of a precataractous type occur in diabetes.

17. Rea, R. L.: Neuro-Ophthalmology, Ed. 2, St. Louis, C. V. Mosby Company, 1941, p. 475.

18. Horner, J. F.: Klin. Monatsbl. Augenh. **11**:488-492, 1873.

19. Duke-Elder,[16] p. 4364.

20. Bellows, John G.: Cataract and Anomalies of the Lens: Growth, Structure, Composition, Metabolism, Disorders and Treatment of the Crystalline Lens, St. Louis, C. V. Mosby Company, 1944, p. 432.

21. Granström, K. O.: Refraktionsveränderungen bei Diabetes mellitus, Acta ophth. **11**:1-160, 1933.

22. O'Brien, C. S., and Allen, J. H.: Ocular Changes in Young Diabetic Patients, J. A. M. A. **120**:190-192 (Sept. 19) 1942.

23. Elschnig, A.: Refraktionsänderungen bei Diabetes mellitus, Med. Klin. **19**:17-18, 1923.

24. Lawrence, R. D.; Oakley, W., and Barne, I. C.: Temporary Lens Changes in Diabetic Coma and Other Dehydrations, Lancet **2**:63-65 (July 18) 1942.

Whatever the cause of these changes, a sudden and unexplained myopia should always suggest the possibility of diabetes, and the ophthalmologist should insist on at least an examination of the urine. It is surprising how often myopia is the first symptom to call the disease to the patient's attention. If it occurs in a known diabetic, an immediate checkup is indicated to ascertain whether or not there has been a relapse. On the other hand, as pointed out by Duke-Elder, a sudden hypermetropic change suggests that treatment may have been undertaken too suddenly or applied too rigorously. The latter situation has been obtained much oftener since the introduction of insulin than before.

The treatment of these transient refractive changes should be directed toward the constitutional disease. Since stabilization of the refractive state of the diabetic may require from two to four weeks, glasses should not be prescribed until this period has elapsed. The patient, moreover, should be warned about the possibility of future sudden changes, and their significance should be explained to him.

Aneurysms of the Conjunctiva.—In 1943 Ballantyne and Loewenstein [25] demonstrated that some of the small spots of blood seen in the retina, which had previously been considered hemorrhages, were actually aneurysms. These authors described them as small, spherical forms attached to the finer, perimacular venules, like berries to twigs.

In 1950 McCulloch and Pashby [26] reported on the examination of 100 diabetics and 50 nondiabetics, in which they found similar aneurysms in the conjunctivas of 55% of the diabetics and 14% of the controls. Two types were seen. The first was a true berry-like aneurysm, such as Ballantyne had described and to which a vessel could or could not be traced. Unlike small hemorrhages, they were purplish, were located at the limbus or toward the fornix, and were deep or superficial. They showed no tendency to absorb, but could be seen unchanged as to size, shape, and position after several months. They were four times as common in diabetic as in normal persons and were found in 62% of diabetics with retinopathy and in 48% of diabetics without retinopathy. They bore no relation to the age of the patient; to the duration of the diabetes; to blood pressure, albuminuria, or hematuria; to the blood cholesterol or nonprotein nitrogen level, or to capillary fragility. This type of aneurysm was regarded by the authors as probably related to the diabetes. The second type appeared as an aneurysmal dilatation of the veins. It was seen anywhere in the conjunctiva, but usually near an area of inflammation. It occurred commonly and was regarded as totally unrelated to the diabetes.

Since conjunctival aneurysms occur in nondiabetic persons, their presence is at most only suggestive of diabetes, and in no way diagnostic. A high incidence of them, however, may indicate a widespread tendency toward diabetes in the general population. Their presence in the conjunctiva suggests, moreover, that they may occur also in most other tissues of the body and that the hyaline changes in the pancreas, the glomerular tufts, and the retina are comparable. McCulloch and Pashby suggest that these hyaline changes may be the cause of diabetes and conclude that the aneurysms found in the capillaries of the conjunctiva are probably

25. Ballantyne, A. J., and Loewenstein, A.: The Pathology of Diabetic Retinopathy, Tr. Ophth. Soc. U. Kingdom **63**:95-119, 1943.

26. McCulloch, C., and Pashby, T. J.: Significance of Conjunctival Aneurysms in Diabetes, Brit. J. Ophth. **34**:495-505 (Aug.) 1950.

related to the diabetes and to the incidence of diabetic retinopathy. They suggest, further, that diabetes may be part of a generalized vascular disease which affects the retina, glomeruli, islets of Langerhans, and conjunctiva.

Changes in the Iris.—Two types of lesions of the iris, iridopathy and rubeosis iridis, may occur as manifestations of diabetes. Iritis and uveitis, on the other hand, have the same incidence, follow the same course, and have the same prognosis whether diabetes exists or not.

Iridopathy: Parsons [27] states that extreme edema of the pigment layer, with formation of numerous small cystic spaces, is common in diabetes. Friedenwald [28] describes a peculiar formation of blisters and cysts in the pigment epithelium of the iris resulting from accumulations of fluid under this layer. The cells of pigment epithelium are filled with glycogen. As a result of liberation of pigment from the epithelial cells, the anterior surface of the lens may be spotted with clumps of pigment; freed pigment is sometimes seen in the aqueous during a cataract extraction. According to Clapp,[29] this pigment clumping in diabetes may be confused with malignant melanoma.

Rubeosis Iridis Diabetica: This was the term given by Salus [30] to signify the presence in diabetics of new-formed vessels in the sphincter region of the anterior surface of the iris. Fralick's gonioscopic studies [31] revealed peripheral synechias and a vascular network covering the corneoscleral trabeculum. According to Duke-Elder,[32] there may be spontaneous disappearance of these vessels. Secondary glaucoma is the rule, and recurrent hyphema is characteristic. In the cases reported in the literature and reviewed by Fralick,[31] retinopathy was usually also present. Lawrence and Levy [33] noted new vessels on both surfaces of the cornea, and Kurz [34] found them on the disk and in the retina.

The visual prognosis for the diabetic patient showing this lesion of the iris is always poor. There is no response to miotics, and antiglaucoma surgery is seldom well tolerated. Agatston [35] states that the condition of the retinal and uveal venous system has a direct bearing on the surgical result. In my experience and in that of Agatston,[35] de Roetth,[36] and others, cyclodiathermy occasionally gives satisfactory results in severe cases by reducing tension and relieving pain. Fralick [31] believes that if the vascular supply to the eyeball is sufficiently deranged, metabolic abnor-

27. Parsons, J. Herbert: Pathology of the Eye, New York, G. P. Putnam's Sons, 1904, Vol. 1, p. 319.

28. Friedenwald, Jonas: The Pathology of the Eye, New York, The Macmillan Company, 1929, p. 211.

29. Clapp, C. A.: Diabetic Iridopathy, Am. J. Ophth. **28**:617-623 (June) 1945.

30. Salus, R.: Rubeosis iridis diabetica, eine bisher unbekannte diabetische Irisveränderung, Med. Klin. **24**:257-258 (Feb. 17) 1928.

31. Fralick, F.: Rubeosis Iridis Diabetica, Am. J. Ophth. **28**:123-139, (Feb.) 1945.

32. Duke-Elder, W. S.: Changes in Refraction in Diabetes Mellitus, Brit. J. Ophth. **9**:167-187, 1925.

33. Lawrence, R. D., and Levy, A. H.: Vascularization of Iris and Cornea in Diabetes, Brit. J. Ophth. **20**:198-201 (April) 1936.

34. Kurz, O.: Zur Rubeosis iridis diabetica, von Graefes Arch. Ophth. **110**:284-302, 1937.

35. Agatston, S. A.: Surgical Eye Complications in Diabetes, Am. J. Ophth. **27**:592-594 (May) 1946.

36. deRoetth, A.: Cyclodiathermy in the Treatment of Glaucoma Due to Rubeosis Iridis Diabetica, Arch. Ophth. **35**:20-23 (Jan.) 1946.

malities will produce a low-grade iritis with new-vessel formation, synechias, and, eventually, hemorrhagic glaucoma. If cyclodiathermy does not control the pain, enucleation is the only recourse.

Changes in the Lens.—It is an old observation that cataract is particularly likely to develop in diabetics. There are, in fact, two types that may occur: (1) the true diabetic cataract, which is seen only rarely, and (2) changes in the lens similar to the senile cataract of elderly nondiabetic persons.

1. True Diabetic Cataract: Typical diabetic cataract, invariably bilateral, is usually seen in young persons with severe diabetes. O'Brien and Allen [22] found the lesion in 14% of 260 diabetic patients under 21 years of age. According to Kirby,[37] it develops in the presence of a high concentration of sugar, and usually when the patient is fairly well, i. e., when he has a sufficient reserve of fluid. It usually appears in patients under 25 and has been seen as early as 11 months. In one patient whom I observed personally the diabetes was diagnosed at the age of 2½ years and the diabetic cataracts were discovered at the age of 6 years. On rare occasions the cataract occurs in older persons, e. g., in a patient aged 33 reported by Kirby,[37] and in a man aged 47, reported by Franceschetti.[38]

The appearance of the true diabetic cataract is characteristic. The opacities are of the snowflake type, start in the subcapsule, and increase rapidly. Kirby [37] tells of one cataract that matured in four days. In addition to the typical lesions, irregular large and small plaques may appear, with fine, dust-like, dirty-brown opacities and rapidly progressive, bluish-white opacities.

Lawrence, Oakley, and Barne [24] point out that temporary changes in the lens can occur in diabetic coma. They are a result of dehydration and disappear when the condition is corrected by isotonic sodium chloride solution replacement. This dehydration factor is believed to be a significant feature of acute forms of the systemic disease in young people.

Bellows and Shoch [39] have shown that lens opacities occur in experimental alloxan diabetes in rabbits. There is loss of glutathione, not caused by the alloxan itself, and this, they believe, is an important factor in the pathogenesis of alloxan-diabetes cataract.

Duke-Elder [40] lists the possible causes of diabetic cataract that have received consideration as follows: (1) presence of sugar in the aqueous and lens, an untenable explanation, since the concentration required to produce a cataract would be incompatible with life; (2) toxic products of metabolism; (3) edema and degeneration of the ciliary epithelium; (4) photochemical activity; (5) endocrine failure; (6) decrease in the permeability of the capsule; (7) influence of acidosis, and (8) hydration of the lens by osmotic influence. Duke-Elder favors the theory of the hydration of the lens.

37. Kirby, D.: Cataract and Diabetes, Arch. Ophth. **9**:966-973 (June) 1933.

38. Franceschetti, A.: Typical Diabetic Cataract in a Man 47 Years Old, Ophthalmologica **101**:241 (April) 1941.

39. Bellows, J. G., and Shoch, D. E.: Allozan Diabetes and the Lens, Am. J. Ophth. **33**:1555-1564 (Oct.) 1950.

40. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 3, St. Louis, C. V. Mosby Company, 1940, p. 3208.

2. Senile Cataract in Diabetes: It is generally agreed that the senile type of cataract appears more frequently in diabetics than in nondiabetics, and at a relatively early age. Kirby [37] found that 64% of diabetic patients of all ages had opacities of some kind, a percentage much higher than that in groups of nondiabetic persons. Of these opacities, 70% were of the senile cortical type. The severity of the disease has no apparent relation to the incidence of the cataract, which in Kirby's group was found more frequently with diabetes of moderate severity. The incidence increased with the duration of the disease, however; for example, 70% of the patients who had had diabetes over five years showed cataractous changes.

It is noteworthy that not all authorities agree with Kirby's finding that cataract is more frequent in diabetic patients. Waite and Beetham [15] and Anderson,[41] for example, came to the conclusion that although punctate opacities and crystalline deposits were commoner in diabetic persons than in the general population, cataract was not.

Cataract Extraction in the Diabetic.—The true diabetic cataract calls for the linear extraction advocated by Barkan [42] and Cordes.[43] The operation avoids trauma to the vascular tissues and thus materially reduces the danger of hemorrhage. If good preoperative dilation of the pupil is not obtained, an iridectomy should be performed through an incision just inside the limbus. No statistics on the surgical prognosis of this procedure were to be found in the literature, but in the three cases in which I operated personally there were no complications and the visual result was satisfactory. The visual prognosis inevitably depends largely on the presence or absence of retinopathy.

In the past cataract extraction in the diabetic has carried a poor prognosis. The complications most dreaded were hemorrhage and infection. In 1911, before the introduction of insulin, Gifford [44] found that 43% of diabetics lost their eyes after cataract extraction as the result of postoperative infection. In a series of 2,123 extractions, Wheeler [45] found hemorrhage in 5% of the nondiabetic patients and in 28.9% of the diabetic patients. He concluded that the presence of diabetes of any degree of severity was an important predisposing factor in the production of these hemorrhages. Both Wright [46] and Kirby [47] concur.

As Duke-Elder [48] points out, the proper use of insulin has changed the prognosis in these cases completely. In reporting the results of cataract extraction in

41. Anderson, C. L.: Contributions to the Statistics of the Diabetic Eye Complications, Acta ophth. **2**:250-256, 1924-1925.

42. Barkan, O.: Procedure for Extraction of Congenital, Soft and Membranous Cataracts, Am. J. Ophth. **15**:117-124 (Feb.) 1932.

43. Cordes, F. C.: Surgery of Congenital Cataracts, Am. J. Ophth. **31**:1073-1084 (Sept.) 1948.

44. Gifford, H.: On Necessity of Extra Care in the Prognosis and Treatment of Diabetic Cataract, Ophth. Rec. **20**:243-245, 1911.

45. Wheeler, J. M.: A Study of Hemorrhage into the Anterior Chamber Subsequent to Operations for Hard Cataract, Tr. Am. Ophth. Soc. **14**:742-752, 1916.

46. Wright, R. E.: Lecture on Cataract Surgery, Am. J. Ophth. **20**:240-252 (March) 1937.

47. Kirby, D.: Prevention and Handling of Complications Arising During and After Cataract Extraction: Some Practical Points, Arch. Ophth. **25**:866-902 (May) 1941.

48. Duke-Elder,[40] p. 3207.

a series of 40 diabetic patients, DeVoe [49] concluded that the presence of diabetes did not greatly alter the prognosis as concerned postoperative bleeding. In my own series the incidence of postoperative bleeding was actually slightly less than in the nondiabetic group. Wright [46] expresses the opinion that certain rigid irises are apt to bleed and that normal ones are not, and, further, that when there are diseased vessels, as in arteriosclerosis and diabetes, the complication of hemorrhage is more likely to occur. DeVoe [49] found that in diabetics the performance of an iridectomy at the time of extraction increased the chance of subsequent hemorrhage by about 20%.

If the retinopathy consists of petechial or small hemorrhages, the surgical risk is good, but engorged veins and large retinal hemorrhages are contraindications to surgery, which should then be delayed or avoided. These pathological changes may be seen if the lens opacities are not too dense, but their presence must be inferred, chiefly through light projection and gross visual-field studies, when ophthalmoscopy is not possible. The condition of the opposite retina, which often is still visible, may be an important guide. If a cataract extraction is to be performed on a diabetic patient in whom neither fundus can be visualized, the patient, or the patient's relatives, should be warned that, although the chance of improving the vision is sufficiently good to warrant operation, the result may be disappointing. Needless to say, operation should not be performed in the presence of vitreous hemorrhages, retinitis proliferans, or detachment of the retina.

If surgery is contemplated, any abnormal elevation of blood pressure should be treated, all foci of infection should be rigidly eliminated, and the diabetes should have been under control for a number of months. The patient should be hospitalized several days before operation, so that dietary conditions and the insulin dosage can be standardized in the hospital where the operation is to be performed. In Agatston's opinion,[35] it is well to reduce the blood sugar by diet or use of insulin to 200 mg. per 100 cc. or less before operation, although insulin should not be given on the day of operation, at least in any large amount, for fear of increasing the danger of hemorrhage.

Diabetic Retinopathy.—The development of diabetic retinopathy is apparently unrelated to the severity of the diabetes (Doggart [4]). It was long believed to be the result of, or associated with, retinal arteriosclerosis and hypertension. Although the two conditions are indeed frequently associated in persons of the older age group, hypertension and the retinopathy of diabetes are separate entities, clinically and histologically (Ballantyne [50]). In both conditions the earliest recognizable lesions are pathological changes in the retinal vessels; but in diabetes these lesions affect primarily the venous side of the retinal vessels, and in hypertension they affect primarily the arterial side.

Of interest in this connection are the cases described by Kimmelstiel and Wilson [9] in which diabetes mellitus, albuminuria, and hypertension were associated clinically and pathologically; this syndrome is the so-called Kimmelstiel-Wilson disease, or intercapillary glomerulosclerosis. The lesion is characterized by the

49. DeVoe, G.: Hemorrhage After Cataract Extraction: Clinical and Experimental Investigation of Its Cause and Treatment, Arch. Ophth. **28**:1069-1096 (Dec.) 1942.

50. Ballantyne, A. J.: Retinal Changes Associated with Diabetes and Hypertension: Comparison and Contrast, Arch. Ophth. **33**:97-105 (Feb.) 1945.

presence of globular hyaline nodules, usually situated near the periphery of the glomerular tuft, by irregular strands of similar hyaline material between and surrounding the glomerular capillaries, and by the presence of enlarged or dilated capillaries in the glomeruli. White and Waskow [14] report that every diabetic child in the Joslin Clinic who had had diabetes for 15 years or more and on whom autopsy was performed showed this lesion of intercapillary glomerulosclerosis. That there is a relation between the vascular system of the retina and that of the rest of the body is brought out by Post and Stickle,[51] who conclude that in juvenile diabetics examination of the retina is a fairly reliable method of determining the amount of renal damage that has been sustained and the condition of the vascular system in general.

Friedenwald [3] found associated renal lesions in 25 of 43 diabetics with retinal microaneurysms and no renal lesions in 33 diabetics without aneurysms. This observation would certainly indicate a close connection between retinal microaneurysms and the Kimmelstiel-Wilson disease. That such aneurysms are also present in nondiabetic patients, however, has been shown by Ashton.[52] He found them in the posterior part of the fundus in 13 of a series of 79 postmortem examinations of nondiabetic patients and concludes that, although they are indeed a valuable diagnostic clue, they are not the unequivocal sign of diabetes that Ballantyne considered them.

In a recent study, Sysi [53] examined 143 pairs of eyes from three series of patients, with arteriosclerosis, nephrosclerosis, and nephritis, respectively, and found microaneurysms in each group. There was no clear difference between the formations occurring in the different diseases. He concludes that the retinal aneurysms were not manifestations of hypertension, arteriosclerosis, retinal disease, hyperglycemia, or pancreatic changes, but were caused, rather, by "disturbances in the central regulation which occur in these diseases." Microaneurysms in the retina, as in the conjunctiva, should thus be considered as suggestive, but not diagnostic of diabetes.

While arteriosclerosis and diabetes are often associated, there are many diabetics in whom there is no evidence of arterial disease. This is particularly true in young patients. Bloch [54] reports among his cases that of a woman aged 26 with diabetes of 15 years' duration and typical diabetic retinopathy. There was no evidence of arterial or renal disease; the blood pressure was 125 systolic and 90 diastolic. O'Brien and Allen [22] found diabetic retinopathy in 4% of a series of young patients without hypertension or arteriosclerosis.

Waite and Beetham,[15] Anderson,[55] Wagener [2] and others have shown that the duration of the diabetes is the most important factor in the production of the retin-

51. Post, L., and Stickle, A. Jr.: Fundus Changes in Juvenile Diabetes, Tr. Am. Ophth. Soc. **48**:191-203, 1950.

52. Ashton, N.: Retinal Micro-Aneurysms in the Non-Diabetic Subject, Brit. J. Ophth. **35**:189-212 (April) 1951.

53. Sysi, R.: Retinal Micro-Aneurysms in Connection with Generalized Diseases Causing Retinopathy, Brit. J. Ophth. **35**:560-567 (Sept.) 1951.

54. Bloch, F.: Retinopathy in Juvenile Diabetes Mellitus, Arch. Ophth. **28**:890-895 (Nov.) 1942.

55. Anderson, G. E.: Problem of Retinitis in the Diabetic Patient, Arch. Ophth. **28**:679-690 (Oct.) 1942.

opathy. Wagener [2] found that 83% of all patients who had had diabetes for over 20 years had diabetic retinopathy, and that the incidence was as high in the cases of controlled diabetes as in the cases of uncontrolled diabetes. Renard and Dhermy [56] evolved a theory to explain the late development of retinopathy. They found that in the course of diabetes of long standing, in addition to upsets in the carbohydrate metabolism, and independent of them, an alteration in the lipotropic function of the pancreas occurs. This induces a disturbance in the lipid metabolism, shown particularly by the fatty changes in and around the vessels, producing the characteristic diabetic retinopathy.

Wagener [2] divides the development of the retinopathy into five stages, which he describes as follows:

1. The earliest sign is the appearance of one or more small capillary aneurysms, which formerly were called "small deep hemorrhages." These were first demon-

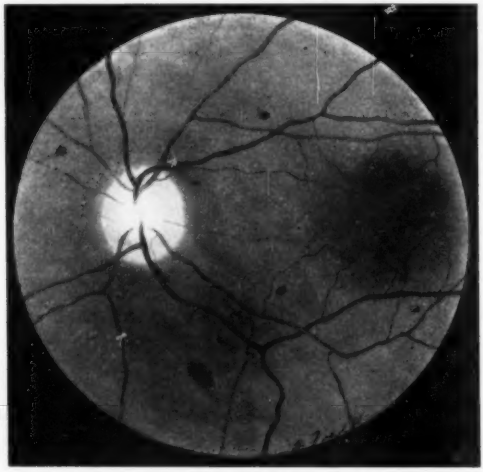

Fig. 1.—Diabetic retinopathy, Stage 1 (Wagener, H. P.; Dry, T. J., and Wilder, R. M.: Retinitis in Diabetes, New England J. Med. **211**:1131-1137 [Dec. 20] 1934), presenting punctate hemorrhages and capillary aneurysms. All the hemorrhages are essentially round.

strated by Ballantyne and Loewenstein,[25] and later by Friedenwald,[57] Ashton,[58] and others. The aneurysmal sacs are often incompletely lined with endothelium (Fig. 1).

2. The next stage is marked by the development of tiny, punctate hemorrhages, chiefly in the macular area. At this stage the exudates begin to appear. This is the central punctate retinitis of Hirschberg (Fig. 2). They are solid, soapy, or waxy looking, yellowish in color, with well-defined, sharply cut edges. At a later

56. Renard, G., and Dhermy, P.: Considérations sur la pathogénie de la rétinite diabétique, Arch. ophth. **10**:170-186, 1950.

57. Friedenwald, J.: A New Approach to Some Problems of Retinal Vascular Disease, Tr. Am. Acad. Ophth. **53**:73-87 (Nov.-Dec.) 1948.

58. Ashton, N.: Vascular Changes in Diabetes with Particular Reference to the Retinal Vessels, Brit. J. Ophth. **33**:407-420 (July) 1949.

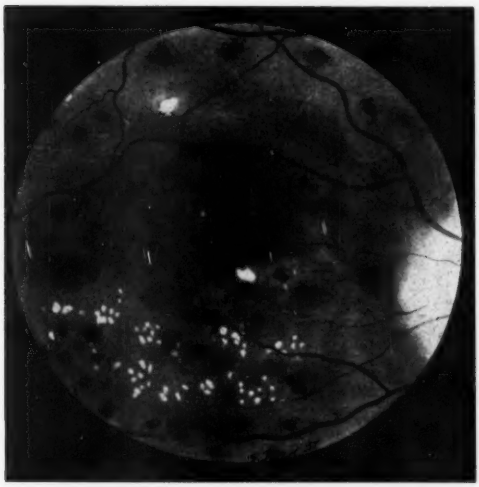

Fig. 2.—Diabetic retinopathy, Stage 2, representing the "central punctate retinitis" of Hirschberg (enlarged tracing of fundus photograph).

Fig. 3.—Late Stage 2, with exudate coalesced into larger masses (enlarged tracing of fundus photograph).

stage they have been described by Doggart [4] as looking like "blobs of white or yellow paint" (Fig. 3). Distributed irregularly in the central area, they frequently form a circle around the macula (Fig. 4). As time goes on these exudates tend to coalesce into lardaceous, irregular masses interspersed with dark-gray pigmentary degeneration.

Duke-Elder stated in a personal discussion that he and Ashton believe that there is a relationship between these lipid, or hard, exudates and the microaneurysms. They believe that the lipids leak out through the wall of the aneurysms, owing to the lack of endothelial lining. This would account for the fact that the aneurysms are often surrounded by a circinate area of these exudates. The same process probably takes place in the kidney in Kimmelstiel-Wilson disease. In support of this

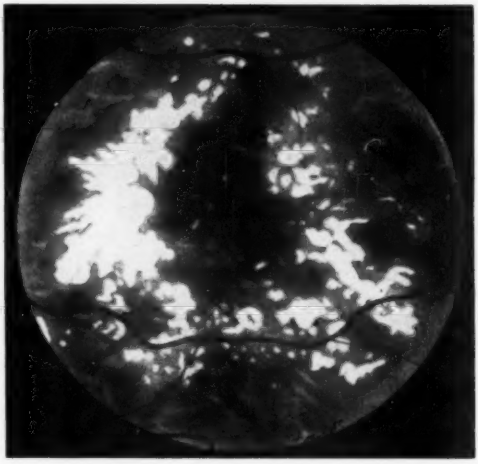

Fig. 4.—Diabetic retinopathy, Stage 2, circinoid form (enlarged form tracing of fundus photograph).

theory is the fact that similar patches of exudates are found around the miliary aneurysms that occasionally occur in young nondiabetic patients affected with a condition related to angiomatosis retinae.

3. In a third stage cotton-wool patches develop, and may indicate the onset of complicating hypertension (Fig. 5) (Koyanagi [59]).

4. Visible changes in the veins occur in a fourth stage, or may in some instances follow Stage 2; when retinopathy occurs in young diabetics this change may even occur as the initial lesion. The veins dilate, develop nodules (beading), and become ensheathed (Fig. 6). It was Nettleship [60] who first observed phenomenon, in

59. Koyanagi, Y.: Spielt die Hypertonie bei der Entstehung der Retinitis diabetica eine wisentliche Rolle? Klin. Monatsbl. Augenh. **94**:353-366 (March) 1935.

60. Nettleship, E.: Hemorrhagic Retinitis in a Patient with Diabetes: Varicose Swellings on a Retinal Vein in the Right Eye, Tr. Ophth. Soc. U. Kingdom **8**:161, 1888.

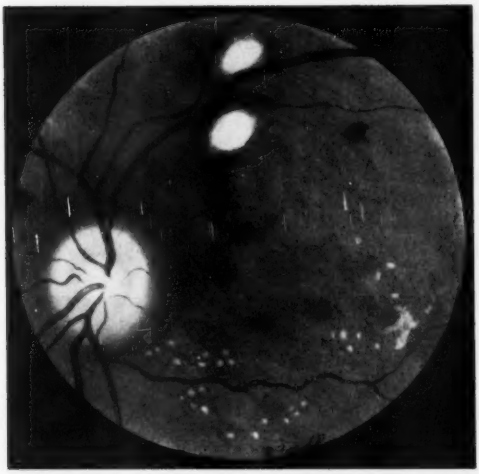

Fig. 5.—Diabetic retinopathy, Stage 3, showing "central punctate retinitis" with cotton-wool patches (enlarged tracing of fundus photograph).

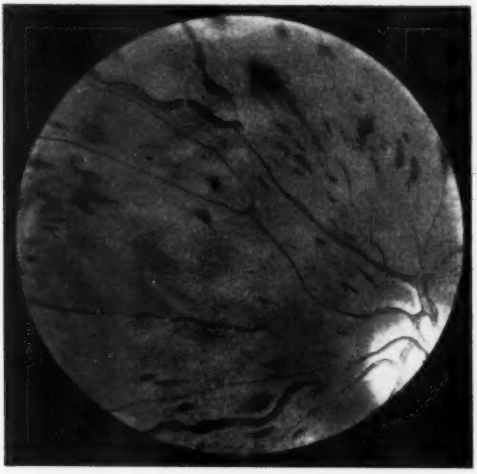

Fig. 6.—Diabetic retinopathy, Stage 4, showing hemorrhages with pronounced disease of the veins (enlarged tracing of fundus photograph).

1888. Since then, Gray [61] and O'Brien and Allen [62] have demonstrated that under the microscope the veins appear sclerosed, the walls showing variations in thickness, hyaline degeneration, areas of endothelial proliferation, and thrombosis. At this stage multiple thrombi may develop, as well as larger hemorrhages, deep and round rather than superficial. The central retinal vein may undergo total obstruction.

5. In the final stage there are recurrent vitreous hemorrhages, which are followed by retinitis proliferans (Fig. 7). According to Gifford,[63] this occurs in only about 1.5% of the patients. It is nevertheless an exceedingly important condition, since it usually leads to detachment of the retina and blindness. Klien [64] has shown that the retinitis proliferans in diabetes originates at or near the disk, with development of a profusion of newly formed blood vessels, which appear in advance of any noticeable amount of connective tissue. These vessels often show a brush-like

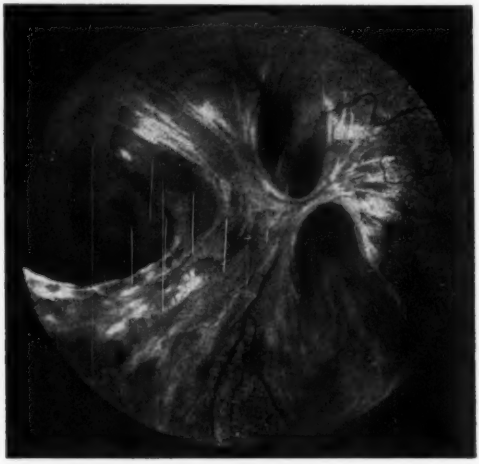

Fig. 7.—Diabetic retinopathy, Stage 5, with preretinal hemorrhage and retinitis proliferans (enlarged tracing of fundus photograph).

arrangement, and the endings in the vitreous form either coil-like convulsions or loops returning to the disk.

In the older diabetic the retinopathy is likely to pass through all five of these stages, but, as noted above, it may start with Stage 4 in young patients who have been kept alive with insulin.

Pathological Features.—A good deal has been written on the histologic features of diabetic retinopathy. Ashton [58] reports postmortem findings in 21 diabetic patients. He observed two types of aneurysms in the retina. One type showed an

61. Gray, W. A.: Retinal Vessel Changes in Diabetes, Tr. Ophth. Soc. U. Kingdom **51**:108-112, 1931.

62. O'Brien, C. S., and Allen, J .H.: Unusual Changes in the Retinal Veins in Diabetes, Arch. Ophth. **24**:742-757 (Oct.) 1940.

63. Gifford, S. R.: Proliferating Retinopathy in Diabetes, Quart. Bull. Northwestern Univ. M. School **17**:252-256, 1943.

64. Klien, B.: Retinitis Proliferans, Arch. Ophth. **20**:426-436 (Sept.) 1938.

afferent and an efferent vessel; the other type arose from one side of the vessel, the opposite side remaining normal. Ballantyne [50] states that the earliest recognizable change takes the form of minute fatty granules in the vascular endothelium, together with swelling of the endothelial cells. He considers the most striking changes in the retinal veins to be expansion, beading, and the formation of loops, coils, and networks; he found predominant histologic changes to be phlebosclerosis and intraretinal and preretinal networks of large, thin-walled vessels. These findings correspond with those of Gartner,[65] O'Brien and Allen,[62] and others. The hemorrhages, as already stated, are characteristically rounded and occur chiefly in the deep layers, particularly in the internuclear layer. Exudates are observed in the deep layers and patches of ganglioform degeneration in the nerve-fiber layer. Renard and Dhermy [56] are of the opinion that the fatty nature of the exudates cannot be explained on the basis of exudation with reabsorption of fibrin by leukocytes; they think, rather, that it points to a lipid infiltration of an ischemic area.

Treatment.—There is no generally accepted treatment for diabetic retinopathy aside from the treatment of the diabetes itself. Moreover, in attempting to evaluate any method of treatment, it must be borne in mind that diabetic retinopathy tends to undergo periodic remissions and exacerbations. According to Friedenwald [3] both punctate hemorrhages and waxy exudates may disappear and then reappear suddenly. Doggart [4] believes that individual hemorrhages can undergo absorption but that exudates seem never to do so, although their appearance may be profoundly changed by coalescence with adjacent foci or by admixture with proliferating pigment.

Although Wagener [2] and others have found the incidence of retinopathy as high in patients with controlled diabetes as in those with uncontrolled diabetes, five recent reports indicate that the careful control of the disease may ward off retinal complications as follows:

1. Root, Sinden and Zanca [66] found that of 282 patients whose diabetes began between the ages of 15 and 30 years and who were observed over a 20-year period, the incidence of retinal lesions, including retinitis proliferans, was much lower in the patients in whom the disease was fairly well controlled than in those in whom it was poorly controlled.

2. Jackson and his co-workers [67] found a highly significant statistical correlation between the incidence of retinopathy and the degree of control of the diabetes; in their report they stress the need for a high level of control in order that serious degenerative changes may be delayed, and sometimes prevented.

3. A most interesting study is reported by Post and Stickle.[51] On the basis of observed fundus changes, they conclude that the degree of control of the diabetes, as well as the ocular findings and the general health, become progressively worse the longer the disease endures; but they find also that the patient who is able to

65. Gartner, S.: Ocular Pathology of Diabetes, Am. J. Ophth. **33**:726-740 (March) 1950.

66. Root, H.; Sinden, R. H., and Zanca, R.: Factors in the Rate of Development of Vascular Lesions in the Kidneys, Retinae and Peripheral Vessels of the Youthful Diabetic, Am. J. Digest. Dis. **17**:179-186 (June) 1950.

67. Jackson, R. L.; Hardin, R. C.; Walker, G. L.; Hendricks, A. B., and Kelly, H. G.: Degenerative Changes in Young Diabetic Patients in Relationship to Level of Control, Pediatrics **5**:959-971 (June) 1950.

control the disease perfectly presents less pathological change than the patient who controls the disease less perfectly or fails to control it altogether.

4. In a study of diabetic retinopathy in young patients, Walker [68] found a close correlation between the development of the retinopathy and the level of control of the disease. While the duration was a significant factor, the correlation of retinal lesions with increased duration of the disease was paralleled by a decrease in the level of control. Walker concludes that in the treatment of juvenile diabetes a high level of control offers the best means of averting or retarding chronic degenerative changes.

5. The most recent study along this line has been made by Sherrill.[69] He reports two groups of patients who had carried out the diabetic routine with "fidelity, patience, and perseverance." One group, of 26 patients, who had acquired diabetes after the age of 18 had had the disease from 20 to 38 years without any visual disturbance. In this group, 4 showed no retinal changes whatever, and the changes in the other 22 were minor only. In a second group of patients, who had acquired the disease before the age of 17, 14 had had it from 20 to 30 years. Two who had had it 30 and 26 years, respectively, revealed no retinal lesions; the rest had no visual disturbances but showed "minor retinal changes."

Radnót and Sipos [70] and others report excellent results with administration of rutin, 20 mg. three times a day, in cases of new-vessel formation and hemorrhage. They believe that it helped to prevent hemorrhages in patients with hypertension, particularly in those with an increased tendency toward abnormal capillary fragility, in their opinion the main cause of retinal hemorrhage in diabetes. Experience with rutin has in general been less favorable. Cometta and Cuendet [71] found it ineffective, and Barnes,[72] in a most convincing article, reports on a series of 32 patients with retinopathy at Joslin's Clinic in Boston to whom he gave rutin for a period of 12 months. Most of them received at least 180 mg. orally a day, and 19 patients took 300 mg. or more a day. In 5 of the 32 patients vitreous hemorrhages, severe enough to impair vision, developed while they were taking rutin, and in no case was the progress of the retinopathy affected. Barnes is of the opinion that while rutin may possibly have value in preventing retinopathy, good control of the diabetes is the best prophylaxis.

Appel [8] thinks that premature aging of the blood vessels, due largely to disturbance of the lipid metabolism, is one of the typical changes of diabetes. Treatment in advanced cases should therefore be directed toward the disease of the vascular system rather than the diabetes itself. For this reason, he advocates sympathectomy.

68. Walker, G. L.: Diabetic Retinopathy in Young Persons, Tr. Am. Ophth. Soc. **48**:677-697, 1950.

69. Sherrill, J. W.: Diabetic Retinitis: The Relationship Between Retinal Degenerative Changes and the Degree of Diabetic Control, Bull. Scripps Metabolic Clinic **2**:1-17 (Oct.) 1951.

70. Radnót, M., and Sipos, L.: Beitrag zur Therapie der Retinitis diabetica, Ophthalmologica **119**:373-377 (June) 1950.

71. Cometta, F., and Cuendet, J. F.: Le traitement des rétinopathies diabétique par la "vasorutine," Bull. mém. Soc. franç. opht. **62**:237-243, 1949.

72. Barnes, R. H.: Capillary Fragility Studies in Diabetes Mellitus and the Use of Rutin in Diabetic Retinitis, Am. J. M. Sc. **219**:368-373 (April) 1950.

The most recent account of a new agent to be tried in the treatment of diabetic retinopathy is contained in a preliminary report by Saskin, Waldman, and Pelner [73]; they treated 28 consecutive, unselected patients with testosterone propionate. The rationale for its use was based on certain observations that have been made on diabetics. In young patients, in whom the diabetes is nearly always a result of actual pancreatic deficiency, insufficiency of the liver often supervenes, and the liver is then unable to inactivate estrogen. The relation of impairment of liver function to the development of arteriosclerosis has been brought out by Rabinowitch.[74] The further fact that the incidence of retinopathy is much higher in females than in males suggests that the male hormone may be useful in diabetic retinopathy. The intramuscular administration of testosterone over a period of from two to six months resulted in the "unequivocal improvement" of more than one-third of the patients and definite improvement in an additional 40%. The authors caution that to prevent any masculinizing effect it is advisable to administer not more than 300 mg. of testosterone propionate a month to women patients. As already stated, this report is a preliminary one only, and further work must be done before testosterone as a therapeutic agent in this condition can be evaluated.

Vitreous Hemorrhage.—In the diabetic, vitreous hemorrhage has a definite tendency to undergo periodic remission and exacerbation. Rutin has been somewhat more successful in controlling it than in controlling retinal hemorrhage. Guyton and Reese [75] used x-ray therapy in two cases of severe diabetic retinopathy with new-formed blood vessels and vitreous hemorrhages. There was an initial improvement in vision, but five months later fresh hemorrhages were still appearing, even though the new-formed vessels were markedly diminished. One patient whom I treated with the 6,000 r recommended by Guyton and Reese has had clearing to such an extent that for the last 18 months she had sufficient vision to get about comfortably and there have been no new vitreous hemorrhages. The retinopathy has progressed gradually, however, and there is more extensive retinitis proliferans. It would seem justifiable to use x-ray therapy in desperate cases of recurrent vitreous hemorrhage, even though the hope of improvement is slight.

Pregnancy and Diabetic Retinopathy.—There seems to be a definite relation of maternal diabetes to congenital defects. Statistics show that the diabetic mother has more than the average liability to abortion, miscarriage, and stillbirth, especially if the disease is not well controlled. Moreover, the incidence of congenital defects and of the mortality of the newborn is twice as high among children of diabetic mothers as it is among children of normal mothers.[4]

Recently, Beetham [76] made some interesting observations on the course of pregnancy in the presence of diabetic retinopathy based on a study of 700 pregnant

73. Saskin, E.; Waldman, S., and Pelner, L.: Diabetic Retinopathy: A New Approach to Therapy with a Steroid Hormone—Testosterone Propionate, Am. J. Ophth. **34**:613-617 (April) 1951.

74. Rabinowitch, I. M.: Relationship Between Impairment of Liver Function and Premature Development of Arteriosclerosis in Diabetes Mellitus, Canad. M. A. J. **58**:547-556 (June) 1948.

75. Guyton, J. and Reese, A.: The Use of X-Ray Therapy for Retinal Diseases Characterized by New Formed Blood Vessels (Eales' Disease, Retinitis Proliferans): Relationship Between Impairment of Liver Function and Premature Development of Arteriosclerosis in Diabetes Mellitus, Tr. Am. Acad. Ophth. **51**:525-542 (May-June) 1947.

76. Beetham, W. P.: Diabetic Retinopathy in Pregnancy, Tr. Am. Ophth. Soc. **48**:205-215, 1950.

women seen in Joslin's clinic. He found that diabetic women without retinopathy did well in pregnancy if the diabetes was meticulously controlled, that the presence of any degree of retinopathy reduced the chance of a successful pregnancy to less than 47%, that the prognosis was very poor if a large amount of hemorrhage was present in the fundus of the diabetic, and that no patient with proliferating retinopathy at the onset of pregnancy survived the pregnancy successfully or maintained useful vision. He believes that diabetic wives should have their children as early as possible, certainly before they have had diabetes 20 years.

From Beetham's observations and my limited experience, it would seem that diabetic patients who show extensive retinal hemorrhages or proliferating retinopathy should not be permitted to sustain a pregnancy.

Lipemia Retinalis.—Lipemia retinalis is an uncommon condition, infrequently reported since Heyl [77] first described it. It has become increasingly rare since the

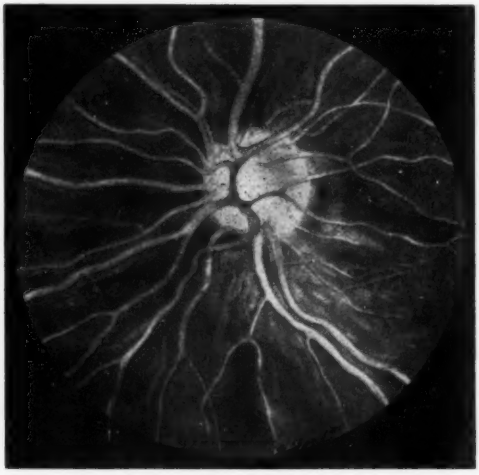

Fig. 8.—Lipemia retinalis in a man aged 29; blood fat, 15%.

advent of insulin. It occurs in severe forms of diabetes, particularly in young men, its production depending on the high neutral-fat content [78] rather than on the height of the total cholesterol. When the blood fat rises above 3.5%, the vessels take on a lighter, salmon color, and above 8% they assume a pinkish-white or entirely white color, as though filled with milk (Fig. 8). This white coloration, as Troncoso [79] points out, is more pronounced in the smaller branches than in the papilla. The fundus preserves its reddish hue. When the blood fat falls below 2.5%, the color returns to normal. Blood drawn from the arm has a chocolate color; after it has stood, a white, cream-like layer forms on the surface.

77. Heyl, A. G.: Intraocular Lipaemia, Tr. Am. Ophth. Soc. **3**:54-66, 1880.

78. Dunphy, E. B.: Ocular Conditions Associated with Idiopathic Hyperlipemia, Am. J. Ophth. **33**:1579-1586 (Oct.) 1950.

79. Troncoso, M. U.: Internal Diseases of the Eye and Atlas of Ophthalmoscopy, Ed. 2, Philadelphia, F. A. Davis Company, 1950, p. 378.

Until 1922, when Wagener [80] reported the first case of lipemia in a nondiabetic patient, the disease was believed to be limited strictly to diabetics. Since then, Dunphy [78] has reported uncovering seven cases of lipemia, including one of his own, in nondiabetic patients.

Insulin therapy is of inestimable value. The lipemia disappears promptly, and the lipids in the blood return quickly to normal. Duke-Elder [81] notes that in all 18 cases of lipemia with one exception, reported before insulin was available, the patient died in coma; since the advent of insulin immediate deaths have been exceptional.

Toxic Amblyopia.—Patients with diabetes are particularly liable to toxic amblyopia. As Doggart [4] and others have pointed out, tobacco is especially dangerous to diabetics and sometimes accounts for a lowering of the visual acuity out of all proportion to the changes in the fundus. Cordes and Harrington [82] found rapid loss of vision of six months' duration in one of their patients who had had severe diabetes for many years. The fundi were essentially normal, but the patient's physician had assumed that the visual loss was due to the diabetes itself.

Nicotinic acid and its derivatives have a strong vasodilator effect. The diabetic is particularly sensitive to the spastic action of nicotine, but Senn [83] found that the very slow intravenous injection of nicotinic acid, up to 50 mg., was effective in tobacco amblyopia. The use of the antispasmotic depropanex® (deproteinated pancreatic extract), combined with oral administration of nicotinic acid, has been effective in our hands. It is well to remember that time may be required for the condition to clear. In one case [82] 2½ months elapsed before the vision returned to 20/30 in each eye. The patient, aged 80, had a severe form of diabetes, but the disease was of recent origin and there was no retinopathy. It was of interest that improvement ceased when the vasodilator injections of sodium nitrite were discontinued.

Optic Nerve Atrophy.—Walsh [84] says that pallor and atrophy of the optic disks in diabetes probably depend largely upon widespread arteriosclerotic changes, which are part of the disease, and that optic neuritis is rarely causative. However, Wolfram [85] reports optic nerve atrophy associated with diabetes in four siblings, whose ages ranged from 7 to 18 years. In discussing these cases, Wagener states that, in view of the gradual onset of loss of vision and its steady progression, a diagnosis of Leber's disease (hereditary optic atrophy) was not justified. The lesion seemed to be bilateral progressive primary optic nerve atrophy. Tyrer [86] reports three siblings between 5 and 18 years of age with severe diabetes and bilateral optic

80. Wagener, H. P.: Lipemia Retinalis: Report of 3 Cases, Am. J. Ophth. **5**:521-525, 1922.

81. Duke-Elder,[40] p. 2744.

82. Cordes, F. C., and Harrington, D.: Toxic Amblyopia Due to Tobacco and Alcohol; Treatment with Vasodilators: Report of 8 Cases, Arch. Ophth. **13**:435-444 (March) 1935.

83. Senn, H. E.: Über die Wirkung von Nicotinsäure und ihren Abköminlingen bei Augenkrankheiten, Praxis **38**:569-570 (June 23) 1949.

84. Walsh, F.: Clinical Neuro-Ophthalmology, Baltimore, Williams & Wilkins Company, 1947, p. 840.

85. Wolfram, D. J.: Diabetes Mellitus and Simple Optic Atrophy Among Siblings: Report of 4 Cases, Proc. Staff Meet., Mayo Clin. **13**:715-718 (Nov. 9) 1938.

86. Tyrer, J.: Case of Infantilism with Goiter, Diabetes Mellitus, Mental Defect and Bilateral Primary Optic Atrophy, M. J. Australia **2**:398-401 (Nov. 13) 1943.

nerve atrophy. The parents were first cousins, and the siblings showed other evidence of neuropathology, so that the role of the diabetes in the development of the atrophy must, of necessity, be in doubt.

Ocular Paralysis.—Paralysis of the extraocular muscles is encountered occasionally in diabetes. According to Duke-Elder,[87] the pathological basis of such an ocular paralysis is not understood, but it is usually ascribed to neuritis of the ocular nerves, comparable to the polyneuritis characteristic of the disease. Waite and Beetham [15] found the condition in 4% of their cases, and Cogan [88] reports observing it in a number of different ocular muscles. Tassman [89] states that the sixth nerve is the one most frequently affected. Complete recovery is the rule, although it may take two or three months.

Ocular Hypotony.—As Moore [90] points out, it has been known for some time that in some cases of diabetic coma there is a striking diminution in the intraocular tension, which is readily appreciable to the touch. It is not due to a corresponding fall in blood pressure, and it has been said to be a result of hyperglycemia. Wolff and de Jongh [91] do not agree with this theory, however, since they observed the same decrease in tension in connection with insulin convulsions (hypoglycemia) and were able to produce the same effect in experimental animals by inducing convulsions with such drugs as strychnine and cocaine. They believe that the substance known as antitonin produced by the convulsions is responsible for the hypotony.

VISUAL PROGNOSIS FOR THE DIABETIC

The ocular complications that occur in diabetes and the possible methods of treating them have been discussed, and the visual prognosis of the diabetic can now be considered.

The introduction of insulin and a better understanding of the dietary requirements of the diabetic have resulted in an increase in his life expectancy. Joslin [92] states that, on the average, diabetics now die at the age of 64, instead of 44, as they did before the introduction of insulin. As a result of this increased longevity, more diabetics are manifesting ocular and other vascular complications. The two most important causes of visual loss are the formation of cataract and the development of retinopathy and its secondary complications.

Cataract is the less serious of these two causes. Diabetic cataract as a specific condition is rare, occurring in only a few of the younger patients; Kumm, Mehlhose, and Obal [93] found it in only 11 of 176 juvenile diabetics. There is no apparent rela-

87. Duke-Elder,[16] p. 4114.

88. Cogan, D.: Neurology of the Ocular Muscles, Springfield, Charles C Thomas, Publisher, 1948, p. 41.

89. Tassman, I. S.: The Eye Manifestations of Internal Diseases, St. Louis, C. V. Mosby Company, 1946, p. 502.

90. Moore, R.: Medical Ophthalmology, Philadelphia, P. Blakiston's Sons & Company, 1922, p. 172.

91. Wolff, L. K., and de Jongh, S. E.: Über eine augenfrucherniedrigende Substanz, Klin. Wchnschr. **4**:344-348, 1925.

92. Joslin, E. P.: Furtherance of Treatment of Diabetes Mellitus, J. A. M. A. **139**:1-7 (Jan. 1) 1949.

93. Kumm, G.; Mehlhose, F., and Obal, A.: Augenbefunde bei jugendlichen Diabetikern, Klin. Monatsbl. Augenh. **116**:482-498, 1950.

tion between the blood sugar level and the development of cataract, and insulin therapy has no favorable effect on it. It has been generally believed that senile cataractous changes are seen more frequently in the diabetic than in the nondiabetic, and Martensson and Palm [94] found changes in the lens in 40% of patients who had had diabetes for at least 15 years. Waite and Beetham,[15] Gifford,[95] and others, however, believe that ordinary changes in the lens are not commoner in the diabetic than in a nondiabetic of the same age. If they are correct in this conclusion, the prognosis for visual loss due to cataract formation is no worse for the diabetic, except for a small percentage of young patients, than for the nondiabetic.

The prognosis for cataract surgery in a diabetic patient was formerly very bad, with loss of the eyeball from infection or hemorrhage a common sequel. Since the introduction of insulin, however, cataract surgery need no longer be feared. The importance of having the disease under the best possible control, of removing any foci of infection, and of stabilizing the diabetic regimen in the hospital several days before operation cannot be overemphasized. Mention of the inadvisability of giving large doses of insulin on the day of operation will also bear repetition. If these precautions are carried out, the surgical prognosis is just as good as it is in the nondiabetic. The visual prognosis depends primarily on the presence or absence of retinopathy. In the true diabetic cataract of young persons, the linear extraction advocated by Barkan [42] and Cordes [43] reduces the danger of hemorrhage to a minimum.

A review of the results of cataract surgery in diabetic patients at the University of California Hospital, in San Francisco, yielded interesting information. In a series of 600 cataract extractions, 71, or approximately 11%, were in diabetic patients. Fifty-four of these patients showed no evidence of retinopathy; in the remaining 17 there were varying degrees of involvement of the fundus. Intracapsular extractions were performed in 73% of the diabetic patients, and extracapsular extractions, in 27%.

The visual results in the diabetic patient compared very favorably with the results in the nondiabetic patient. In 74% of the nondiabetic patients postoperative vision of 20/30 or better was obtained; of the diabetic patients without retinopathy, 70% had equally successful results. The visual results were thus only slightly better in the nondiabetic than in the diabetic without retinopathy. Of the diabetics with varying degrees of retinopathy, only 54% had 20/50 vision or better; 38% had less than 20/200; none had 20/20.

In this series any evidence of blood in the anterior chamber, even a trace, was recorded as hemorrhage. In the diabetic patients it occurred in 19%; in the hypertensive patients, in 21%, and in the arteriosclerotic patients, in 26%. In these three groups, then, the incidence of hemorrhage varied only slightly, and the diabetic group showed the least amount. In none of the diabetic patients was there any evidence of postoperative infection.

94. Martensson, J., and Palm, E.: Ocular Findings in Long-Standing Diabetes Mellitus: Report of 163 Cases of at Least 15 Years' Duration, Acta med. scandinav., Supp. 246, pp. 154-165, 1950.

95. Gifford, H.: Diabetes: Panel Discussion, J. Omaha Mid-West Clin. Soc. **10**:96-98 (Aug.) 1949.

By far the most important cause of permanent loss of vision in the diabetic is retinopathy and its complications, vitreous hemorrhage, and associated retinitis proliferans. In diabetes the life expectancy is greater than it is in simple arteriosclerosis, and much greater than it is in renal disease. The existence of diabetic retinopathy adds little or nothing to the gravity of the general prognosis; it is merely part of the vascular disease associated with diabetes and expressed by retinal hemorrhages and varying degrees of albuminuria, or hypertension, or both.

From the standpoint of vision, however, the ultimate prognosis of diabetic retinopathy is poor, for, in spite of treatment, the changes tend to be progressive. As has been amply covered in earlier sections of this paper, the development of retinopathy is related to the duration of the diabetes rather than to the age of the patient, the severity of the disease, or the method of treatment; and the general increase of life expectancy in diabetes has been parelleled by an increase in its degenerative vascular complications.

There is very little in the literature on the amount of visual handicap resulting from diabetic retinopathy, and very little on the relation of visual acuity to the duration of the diabetes. Gifford,[95] in 1949, in reviewing his records for the previous two years, found 25 cases of diabetic retinopathy; the duration of the diabetes varied from 2 to 20 years, with an average of 10 years. Twelve patients, or nearly 50%, had lost the ability to read ordinary newsprint, their visual acuities being less than 20/70 in the better eye; 6 had less than 20/200 vision, and 4, less than 10/200 vision. These last four patients (8%), although retaining a little vision, had lost their independence and were for all practical purposes blind. This series, while very small, shows that the degree of visual loss in cases of diabetic retinopathy can be very high.

The visual prognosis in diabetic retinopathy is best considered under two general headings: (1) the prognosis for the juvenile diabetic and (2) the prognosis for the adult diabetic.

The Juvenile Diabetic.—White and Waskow [14] found vascular disease in 93% of 220 juveniles surviving 20 or more years of diabetes; in this group the onset of the disease had occurred before the age of 15 years. Chute [96] found retinitis in all of 24 juvenile diabetics who had survived 20 years. The number of young diabetics who have been kept alive on insulin for 20 years or more is increasing, so that more patients with diabetes dating from childhood are now being seen with retinopathy. As has already been stated, in this group the retinopathy is more apt to start with "Stage 4," is severer, and often progresses rapidly to retinitis proliferans. Thus, the visual prognosis of the patient who acquires his diabetes in childhood is poor once middle life has been reached.

This increased number of instances of retinopathy in young diabetics who have had the disease for 20 years or more has stimulated new interest in juvenile diabetics, however, and the recent studies referred to previously indicate that a person with perfectly controlled diabetes may go through a normal life span with a minimum of degenerative changes resulting from the diabetes. This should be a stimulus to both

96. Chute, A. L.: Survey of Patients with Juvenile Diabetes Mellitus, Am. J. Dis. Child. **75**:1-10 (Jan.) 1948.

patient and physician to maintain a normal blood chemistry and to treat both minor and major complications early and adequately, so that the visual prognosis of juvenile diabetes need not be so bad as it has been in the past.

The Adult Diabetic.—Among the patients who acquired diabetes in adult life, Wagener [2] found that 73% of those who had had the disease for more than 20 years had retinopathy. Dolger [97] studied 200 patients with diabetes of 25 years' duration and found all of them with vascular damage and albuminuria or hypertension. There is thus no doubt that the visual prognosis is poor for patients surviving the disease for 25 years, but the number who live so long is relatively small, since in the adult the disease occurs most commonly between 40 and 70 years of age. Of the entire adult group, according to Martensson and Palm,[94] 60% do not have retinopathy. In the adult, moreover, there is a greater tendency for the retinopathy to develop slowly, and many patients observed over a number of years show nothing more in the fundus than the early vascular changes, i. e., microaneurysms and small hemorrhages. It is thus apparent that in the adult the older the patient is at the time of onset of the disease, the better his visual prognosis, and that if he lives more than 20 years after the onset the visual prognosis is not good.

The conclusions of Dolger [97] and others that there is vascular damage in virtually 100% of cases in which the diabetes is of 25 years' duration is not shared by Joslin.[92] He believes that the statistically high rate of damage is in part the result of poor treatment, and he cites the cases of seven patients [98] who have lived 25 years with diabetes and have shown no signs of degenerative changes in the eyes or vascular system elsewhere. Robson [99] cites a case of diabetes of 30 years' duration, with no complications. Sherrill [69] reported a patient with onset of diabetes at the age of 14 who had had the disease for 30 years, and another with onset at the age of 16 whose disease was of 26 years' duration. Neither showed any retinal lesions. In the adult group there were patients who had had diabetes for 21, 22, and 29 years who revealed no retinal lesions. In all these patients, young and old, there had been constant adequate control of the diabetes.

CONCLUSIONS

On the basis of personal experience and a review of the literature, it would seem justifiable to draw the following conclusions regarding the visual prognosis for the diabetic patient:

1. The visual prognosis of rubeosis iridis is very poor.

2. Extraction of a true diabetic cataract, or of a senile cataract in a diabetic patient, offers a good prognosis for vision provided there is no retinopathy. It is important that all foci of infection be cleared up before operation, that the diabetes be controlled in the hospital for several days before operation, and that insulin not be given in large amounts on the day of operation.

3. The young diabetic who survives 20 years is very likely to have a severe form of diabetic retinopathy with loss of vision, irrespective of the level of control at which the disease has been maintained.

97. Dolger, H.: Clinical Evolution of Vascular Damage in Diabetes Mellitus, J. A. M. A. **134**:1289-1291 (Aug. 16) 1947.

98. Joslin, Elliott: Diabetes (Correspondence), J. A. M. A. **139**:603 (Feb. 26) 1949.

99. Robson, H., in Discussion on Diabetes, Bull. Univ. Calif. M. Center **1**:415 (Feb.) 1950.

4. Since the disease in the adult occurs as a rule between the ages of 40 and 70 years, only about 40% of patients with diabetes acquired in adult life have retinopathy.

5. The adult diabetic who survives 20 years with the disease will almost certainly have retinopathy, but the retinal process will usually progress more slowly than it does in the juvenile diabetic.

6. While almost all observers have agreed that the treatment of diabetes has no effect on the development of retinopathy, recent scattered reports indicate that if present-day methods of control are carried out meticulously the prognosis in the future may not be as black as it is now generally believed to be.

7. In view of the devastating effect of loss of vision, the physician managing a diabetic patient carries a grave responsibility to leave no stone unturned to assure the best possible control of the diabetes, and thus perhaps prevent visual loss. To this end, he should take full advantage of the accumulated knowledge available to him.

PAPILLEDEMA AS THE OUTSTANDING SIGN IN MENINGEAL HYDROPS

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SINCE the presenting signs and symptoms of meningeal hydrops are oftenest blurred vision, headache, and papilledema, it is important that ophthalmologists be acquainted with this entity. Accordingly, this report will be concerned with a description of meningeal hydrops and an analysis of seven cases from the ophthalmological and neurological services of the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital.

Meningeal hydrops is a clinical entity in which increased intracranial pressure is present without an expanding intracranial lesion or other obvious cause. With the usual symptoms and signs of headache, blurred vision, and bilateral papilledema, the cerebrospinal fluid is clear, and there is usually no abnormality in cells or protein, although the pressure is elevated. However, there may be exceptions, as reported by Ray and Dunbar [1] and as noted in the case reports of Davidoff and Dyke [2] and Dandy,[3] in which a relatively slight increase in the cerebrospinal fluid cell count or protein occurred.

The following terms have been used in the literature to connote this condition: (*a*) pseudotumor cerebri,[4] (*b*) otitic hydrocephalus,[5] (*c*) intracranial hypertension of unknown cause,[6] (*d*) intracranial pressure without brain tumor,[3] (*e*) toxic hydrocephalus,[7] (*f*) serous meningitis,[2] (*g*) meningeal hydrops,[8] and (*h*) arachnoiditis (Cushing).[4]

Passot, in 1912, first used the name of meningeal hydrops for this syndrome, which he found sometimes followed simple otitis media. This name best fits the findings, since it is not a meningitis, nor is it always related to otitis media or known toxin-producing factors.

From the Massachusetts Eye and Ear Infirmary, and the Howe Laboratory of Ophthalmology, Harvard Medical School.

1. Ray, B. S., and Dunbar, H. S.: Thrombosis of the Dural Venous Sinuses as a Cause of "Pseudotumor Cerebri," Ann. Surg. **134**:376-386, 1951.

2. Davidoff, L. M., and Dyke, C. G.: Presentation of a Series of Cases of Serous Meningitis, J. Nerv. & Ment. Dis. **83**:700-705, 1935.

3. Dandy, W. E.: Intracranial Pressure Without Brain Tumor, Ann. Surg. **106**:492-513, 1937.

4. Frazier, C. H.: Cerebral Pseudotumors, Arch. Neurol. & Psychiat. **24**:1117-1132, 1930.

5. Symonds, C. P.: Otitic Hydrocephalus, Brain **54**:55-71, 1931.

6. Sahs, A. L., and Hyndman, O. R.: Intracranial Hypertension of Unknown Cause: Cerebral Edema, Arch. Surg. **38**:428-442, 1939.

7. McAlpine, D.: Toxic Hydrocephalus, Brain **60**:180-203, 1937.

8. Passot, R.: Méningites et états méningés aseptiques d'origines otique, Thesis, Paris, G. Steinheil, 1913.

The onset of symptoms is usually not sudden; in fact, cases are occasionally found during a routine ophthalmologic examination without any specific complaints having been present. On the other hand, it may follow an acute illness of short duration, as reported by McAlpine.[7] Symptoms referable to increased intracranial pressure, other than headache and blurred vision, may occur, e. g., dizziness, drowsiness, diplopia, nausea, and vomiting.[3] Outstanding is the apparent well-being of these patients, and there is rarely any abnormality of the blood, urine, or temperature. Ray and Dunbar [1] reported that convulsions may occur (if there is thrombosis involving the cerebral veins), and they also noted fever in some cases in which an infected thrombus was presumed to exist.

The papilledema is bilateral and in most cases symmetrical. Davidoff and Dyke [2] found no case with less than 2 D. of papilledema. There may or may not be peripapillary hemorrhages. Even though complaints of blurred vision are frequent, visual acuity is usually good; however, visual-field examinations most frequently reveal enlarged blind spots, and a number of authors have reported generalized constriction of the visual fields.[9]

The remainder of the neurological examination shows no characteristic abnormalities; but there may be isolated signs, such as hyperactive deep reflexes, the Romberg sign, slight ataxia, and the Babinski sign.[3] The ventriculogram reveals no asymmetry or dilatation of the ventricles, despite the term hydrops; in fact, the ventricles often appear smaller than normal. Dural-sinus venography has been done in a small number of cases [1] thus far and has shown obstruction in the superior longitudinal or the lateral sinuses.

The course of meningeal hydrops is variable. The duration may be several weeks or years, even after decompression has been performed. There are marked fluctuations in the intracranial pressure during this period, and normal readings may be obtained intermittently.[3]

The diagnosis is made on the basis of the (1) history; (2) the paucity of neurological signs, especially those having localizing significance; (3) the presence of clear cerebrospinal fluid, usually free of cells, without increased protein but under increased pressure, as indicated by papilledema and manometric measurements; (4) exclusion of intracranial space-filling lesions by ventriculography, and (5) in some instances by dural-sinus venography. An eventual, but usually uncomplicated, recovery is additional evidence in support of this diagnosis.

Treatment in this condition has varied from simple observation to cranial decompression, and Ray and Dunbar [1] obtained improvement in one of their cases only after the superior longitudinal sinus was opened and a clot removed. The same workers believed that heparin and bishydroxycoumarin were of benefit in two patients who had evidence of thrombosis. Repeated lumbar punctures with cautious reduction of the cerebrospinal fluid pressure are done by some. In addition, dehydration measures, such as limitation of the fluid intake, use of saline cathartics, and intravenous administration of hypertonic glucose solution, may be employed.[2]

The pathogenesis of meningeal hydrops is still not settled. Symonds [10] suggested that obstruction of the lateral venous sinus due to thrombophlebitis, such as that

9. Davidoff, L. M., and Dyke, C. G.: Hypertensive Meningeal Hydrops: A Syndrome Frequently Following Infection in the Middle Ear or Elsewhere in the Body, Am. J. Ophth. **20**:908-927, 1937. Sahs and Hyndman.[6] Dandy.[3]

10. Symonds, C. P.: Hydrocephalic and Focal Cerebral Symptoms in Relation to Thrombophlebitis of the Dural Sinuses and Cerebral Veins, Brain **60**:531-550, 1937.

following otitis media, might be the initiating cause in his case. He thought that the thrombus beginning in the lateral sinus could spread by extension to the superior longitudinal sinus and thus interfere with absorption of the cerebrospinal fluid through the arachnoid villi. The patient improved in a number of months. Symonds [10] also cited a case reported by Bailey and Haas in which the clinical picture of meningeal hydrops developed after otitis media. This patient died of cardiac disease, and autopsy revealed thrombi from the right sigmoid sinus to the torcular Herophili and in the posterior two-thirds of the superior longitudinal sinus. Of 12 patients with this entity, which he saw and called "otitic hydrocephalus," 5 had lateral sinus thrombosis at the time of the mastoid operation. He cited a report by Nielsen and Courville on postmortem observations in 43 cases of lateral sinus thrombosis associated with otitis media, in 6 of which the thrombus had extended into the superior longitudinal sinus. It was not stated whether the typical clinical picture had occurred in the latter cases. Symonds stated that if the progression of the thrombus is slow the superior longitudinal sinus can be thrombosed without producing symptoms, provided adequate collateral venous drainage is established for the cerebral cortex. Even when symptoms are produced, the clot is eventually organized, and recanalization of the thrombus takes place, thus paving the way for eventual recovery.

Davidoff [11] agreed with Symonds' impression that the causative factor is incomplete thrombosis of the dural sinuses, preventing proper absorption of cerebrospinal fluid; but in the one pathological report which he presented,[2] that of a patient who died of a cause other than serous meningitis three months after having had this condition there was no evidence of inflammation or thrombosis of the central nervous system. Only questionable evidence of cerebral edema was found.

Dandy,[3] on the other hand, did not believe there was obstruction in the venous sinuses, since the Queckenstedt maneuver revealed no block and the intracranial pressure fluctuated greatly within a few minutes. He observed the latter phenomenon in his cases with subtemporal decompression, and this led him to believe that there was a variation in the intracranial vascular bed under vasomotor control. He observed this phenomenon also with changes in the emotional state. McAlpine [7] expressed the opinion that there existed a true cerebral edema due to circulating toxins, causing an overproduction of cerebrospinal fluid. He drew this conclusion from the history of a preceding infection of the upper respiratory tract in his cases.

Ford and Murphy [12] pointed out that if thrombosis involves the venous sinuses extensively enough there develop massive edema and hemorrhage of the brain. They reported two cases of this syndrome in which autopsy was performed. In the first case, that of a woman aged 26, death was due to herniation of the brain following a decompression. Autopsy revealed congestion and edema of the brain, a partially organized thrombus extending the entire length of the superior longitudinal sinus and involving its collaterals, the torcular Herophili and the left lateral sinus. The second case was that of a girl of 7 years who recovered from this syndrome after a subtemporal decompression. Two years later the child had

11. Davidoff, cited by Yaskin, J. C.; Groff, R. A., and Shenkin, H. A.: Severe Papilledema in Diffuse Infections of the Nervous System, Tr. Am. Neurol. A. **72**:21-26, 1947.

12. Ford, F. R., and Murphy, E. L.: Increased Intracranial Pressure: Clinical Analysis of Causes and Characteristics of Several Types, Bull. Johns Hopkins Hosp. **64**:369-395, 1939.

mastoiditis, which developed into fatal meningitis. Autopsy revealed that the left lateral sinus was replaced by a fibrous cord, the thrombosis antedating, in the pathologist's opinion, the present illness. These authors suggested that the following factors might be operative in such cases of thrombosis of the venous sinuses: (1) edema of the brain; (2) hemorrhage into the brain, in some cases; (3) distention of the intracranial venous channels; (4) diminished absorption of cerebrospinal fluid, due to obstruction of veins into which it is normally absorbed, and (5) questionable increase in production of cerebrospinal fluid, due to congestion of the choroid plexuses.

A recent report by Ray and Dunbar [1] lends weight to the concept of thrombosis of the superior longitudinal and/or the lateral venous sinuses as being the mechanism involved in producing this entity. These authors made venographic studies of the dural sinus with iodopyracet (diodrast®) in four patients with this condition and obtained evidence of a block in the superior longitudinal or the lateral sinuses in all. The venous pressure in the superior longitudinal sinus was elevated in all four patients, the normal being 100 to 150 mm. of isotonic sodium chloride solution.

REPORT OF CASES

Seven cases representing this syndrome are presented. One patient died while in the hospital, and the autopsy findings are included in the report.

CASE 1.—A. B., a woman aged 29, was admitted on Nov. 4, 1949, with a chief complaint of headaches for 10 months. She had had frontal headaches, which were worse on the right and varied in intensity. In September, 1949, she had blurring of vision and diplopia. There were also unsteadiness of gait and unsteadiness of the left hand, with a tendency to drop objects carried in that hand.

Vision was 20/20 in each eye. There was papilledema of 3 to 4 D. in each eye, with enlarged blind spots and peripapillary hemorrhages.

Roentgenograms of the skull and the electroencephalogram revealed no abnormality.

On Nov. 7 a ventriculogram revealed that the initial pressure was 220 mm. of water and that the ventricles were small and in the midline.

Lumbar puncture on Nov. 11 revealed an initial pressure of 235 mm. of water with a final pressure of 150 mm. of water and 10 white cells per cubic millimeter. On Nov. 13 the initial pressure was 235 mm. of water with a final pressure of 150 mm. of water, and 474 red blood cells and 255 white cells per cubic millimeter, 70% monocytes, and 30% polymorphonuclear leucocytes. On Nov. 17 the initial pressure was 260 mm. of water with a final pressure of 180 mm. of water, and 90 red blood cells and 10 monocytes per cubic millimeter. On Nov. 19 the initial pressure was 110 mm. of water.

On Nov. 29 the blind spots were reduced in size and the papilledema had decreased to 2 D. in each eye.

The patient was followed in the outpatient department with repeated lumbar punctures. These at first showed an elevated pressure; then the pressure became normal, symptoms decreased, and the papilledema subsided. She was last seen on Aug. 29, 1951, at which time vision was 20/20 in each eye, the visual fields were normal, and the disk margins were blurred, with no elevation.

CASE 2.—G. N., a man aged 43, was admitted on March 31, 1951, for blurred vision, of five days' duration. The patient stated that three weeks prior to admission he had "flu" and began to have frequent frontal headaches, which still occurred. Five days prior to admission he noted progressive blurring of vision in both eyes, so that it was difficult to read newsprint, and he also stated that he had been unusually nervous.

He had had a nervous breakdown in 1939, for which he was treated in a mental hospital for 3½ months.

Vision was 20/70 in each eye, and 2 D. of papilledema was present bilaterally. Examination of the visual fields showed enlarged blind spots and a lower nasal quadrant defect in the left eye only.

Roentgenograms of the skull revealed no abnormality. The electroencephalogram was mildly abnormal, with scattered slow waves, a bit more pronounced on the right.

Lumbar puncture revealed an initial pressure of 150 to 240 mm. of water with a final pressure of 160 mm. of water, 58 mg. of protein per 100 cc. and no white cells. Lumbar puncture with repeated continuous readings over two hours while the patient was under sedation, and even sleeping, showed that the pressure fluctuated between 160 and 310 mm. of water. The cerebrospinal fluid on several occasions was reported to contain 40 to 45 mg. of protein per 100 cc. The ventriculogram showed an initial pressure of 240 mm. of water, with a normal appearance of the ventricles in roentgenograms.

In daily lumbar punctures the pressure was observed to vary from 0 to 550 mm. of water, and when the pressure had been normal for two consecutive days the patient was discharged. The papilledema remained unchanged. He continued to return to the outpatient department for weekly lumbar punctures, which frequently showed an increased pressure. On May 24, 1951, the papilledema was decreased but was still present. Vision had improved to almost 20/30 in each eye.

CASE 3.—E. S., a woman aged 26, was admitted on June 23, 1951, for mild bifrontal headaches, which had come on in the afternoon or evening during the past month.

The patient was referred to the hospital by her local ophthalmologist, who on routine examination found papilledema in both eyes. There had been tinnitus in both ears intermittently for a "long time." The rest of the history was noncontributory.

Examination revealed papilledema of 2½ D. in the right eye and 3½ D. in the left eye, with no hemorrhages. The fields were normal, and the blind spots were not enlarged. Vision was 18/20 for distance and 14/14 for near in each eye.

Roentgenograms of the skull and chest revealed no abnormality. The electroencephalogram and ventriculogram indicated a normal condition.

Lumbar puncture showed an initial pressure of 270 mm. of water with a final pressure of 250 mm. of water. The protein content, serologic reactions, colloidal gold curve, and cell content were normal.

The third day following the ventriculographic study a lumbar puncture revealed an initial pressure of 90 mm. of water with a final pressure of 80 mm. of water and 50 mg. of protein per 100 cc., and a subsequent lumbar puncture showed an initial pressure of 85 mm. of water with a final pressure of 65 mm. of water.

The referring ophthalmologist informed us that the patient was asymptomatic, and her eyes appeared unchanged in September, 1951.

In December, 1951, she was asymptomatic, and the papilledema was reported to be decreasing.

CASE 4.—J. B., a girl aged 16, was admitted on Sept. 20, 1950, for headache and dizziness, of two weeks' duration.

The patient had had occipital headache, radiating to the frontoparietal region, which was worse on the left. There had been projectile vomiting and diplopia at the onset, and on admission vision was blurred. She had experienced tinnitus of both ears and vertigo on one occasion. She also complained of stiffness of the neck.

Physical examination showed some stiffness of the neck, staggering gait, with swaying to the left, and weakness of the left arm and leg. There was impairment in performance of rapid rotating alternating movements, more pronounced on the left. Her reflexes were hyperactive, and the left plantar response was equivocal. Position sense was diminished in the toes and fingers on the left side. Vision was 18/40 in the right eye and 18/50 in the left eye, with papilledema of 1½ to 2 D. bilaterally. There was sixth-nerve paresis, with nystagmus on lateral gaze to either side. The visual fields were notably constricted peripherally.

On Sept. 22 a ventriculogram revealed a normal state. Lumbar puncture showed an initial pressure of 275 mm. of water; 3,300 red blood cells, 10% polymorphonuclear leucocytes, and 5% lymphocytes per cubic millimeter, and 41 mg. of protein per 100 cc. Roentgenograms of the skull revealed no abnormality. Lumbar puncture on Sept. 23 showed an initial pressure

of 320 mm. of water with a final pressure of 90 mm. of water. On Sept. 24 the initial pressure was 275 mm. and the final pressure 100 mm. of water. Lumbar puncture repeated on the same day revealed a normal pressure.

The electroencephalogram suggested mild abnormality, without focus or specificity.

On Oct. 5 a lumbar puncture showed an initial pressure of 95 mm. of water, with 6 lymphocytes per cubic millimeter.

On Sept. 23 visual acuity was found to be reduced to counting fingers at 2 ft. (60 cm.) in the right eye, and at 6 in. (15 cm.) in the left eye. This determination followed ventriculography, and it was thought that the reduction was a postventriculography effect.

On Sept. 25 vision was 18/40 in each eye, and the papilledema was less pronounced.

The weakness and cerebellar signs on the left had subsided, and at the time of discharge, on Oct. 6, there was no papilledema.

The patient was readmitted on Nov. 30, complaining of a continuous dull occipital headache. She had also had a return of unsteadiness of gait. Vision had remained fairly good. Examination showed slight weakness of the left extremities and slight impairment in performance of the cerebellar tests on the left. Position holding was slightly impaired on the left. Vision was 16/20 in each eye, and there was no papilledema or weakness of the extraocular muscles. The visual fields were normal. Lumbar puncture showed an initial pressure of 110 mm. of water, 2 lymphocytes per cubic millimeter, and 60 mg. of protein per 100 cc. A repeated lumbar puncture showed an initial pressure of 90 mm. of water, 46 mg. of protein per 100 cc., and no cells. The electroencephalogram was normal, and guinea-pig inoculation for tuberculosis gave negative results.

The patient's symptoms disappeared when she was befriended by another young girl in the ward; therefore it was decided to observe her after she had been in a different environment for a while. When she was seen on April 21, 1951, after having been placed in a foster home, she had less headache and normal neurological state.

CASE 5.—A. J. G., a woman aged 40, was admitted on March 19, 1943, for headaches and visual difficulty, of two years' duration. The headache had had a gradual onset, becoming progressively worse. There was blurring of vision in the right eye eight months, and in the left eye five months, prior to admission. There also had been tinnitus and decreased hearing in the right ear.

Physical examination revealed bilateral papilledema, which was moderate to marked in degree. Vision was 20/20 in each eye, with enlarged blind spots and peripheral constriction of the fields.

Roentgenograms of the skull and chest revealed no abnormality. In two ventriculograms filling was inadequate; therefore a pneumoencephalographic study was performed, revealing no evidence of hydrocephalus or brain tumor. The ventricular fluid was clear and colorless, with 14 mg. of protein per 100 cc. The electroencephalogram was within normal limits. The first lumbar puncture following pneumoencephalography showed an initial pressure of 250 mm. of water with a final pressure of 150 mm. of water, 22 mg. of protein per 100 cc., and 10 polymorphonuclear leucocytes, 135 lymphocytes, and 55 monocytes per cubic millimeter. A repeated lumbar puncture showed an initial pressure of 105 mm. of water.

The patient's symptoms gradually diminished, and vision became clear, although the visual fields showed bizarre changing patterns. The headaches decreased until April 26, 1945, when the blind spots were normal in size. On Feb. 21, 1946, the ophthalmologist reported a postpapilledema optic nerve atrophy, although vision was still 20/30 in the right eye and 20/20 in the left eye. At that time the patient had no complaints, and her neurological status was normal.

She was asymptomatic when examined on Sept. 8, 1951. Vision was good, although there was pallor of both disks with blurred margins. There was also an inferior nasal constriction of the peripheral field of the left eye.

CASE 6.—C. M. T., a woman aged 32, was admitted on April 6, 1951, for intermittent blurring of vision and vertigo on straightening up for the past eight months. She had also noted a gain in weight, with increased appetite, polyuria, loss of libido, and a change in the quantity of her menstrual flow.

Between the ages of 19 and 21 she had had intermittent headaches and blurring of vision. There was a history of mental illness, for which she had been given electric shock therapy one year prior to onset of the present illness.

There was papilledema of 2 to 3 D. in each eye, and the blind spots appeared enlarged. Vision was 20/20 in each eye.

The electroencephalogram was normal. The ventriculogram showed a normal condition except for the initial pressure of 240 mm. of water. The cerebrospinal fluid contained 40 mg. of protein per 100 cc., and 34,000 red blood cells, 22 lymphocytes, and 3 polymorphonuclear leucocytes per cubic millimeter.

While the patient was in the hospital, a series of lumbar punctures were done, with the following findings:

(*a*) Initial pressure 540 mm., final pressure 240 mm.; fluid pink.

(*b*) Initial pressure 40 mm.; fluid pink, slightly xanthochromic.

(*c*) Initial pressure 230 to 240 mm., final pressure 130 mm.; fluid straw-colored.

(*d*) Initial pressure 140 to 150 mm., final pressure 80 mm.; fluid straw-colored.

(*e*) Initial pressure 190 mm., final pressure 85 mm.

At the time of discharge the papilledema was unchanged. The patient returned to the outpatient department for repeated lumbar punctures, which revealed pressures varying from normal to abnormal. The last lumbar puncture, done on July 12, 1951, was normal. No papilledema was present, and visual fields, determined on June 15, 1951, showed only enlarged blind spots.

CASE 7.—C. B., aged 43, was admitted on Nov. 1, 1950, for loss of vision in both eyes. There had been a painless loss of vision since July, 1950.

The blood pressure was 190/124. The patient did not appear to be ill and was ambulatory. There was no light perception in the right eye, and vision was 20/40 in the left eye. Papilledema of 3½ D. with venous congestion and prominent peripapillary hemorrhages was present in the right eye. Papilledema of 2½ D., with fewer peripapillary hemorrhages was present in the left eye. Nasal-field loss was found in the left eye by confrontation tests.

Roentgenograms of the skull showed osteoporosis and erosion of the posterior clinoid processes. The ventriculogram revealed a normal condition.

Lumbar puncture done on Nov. 6, 1950, showed an initial pressure of 470 mm. of water with a final pressure of 430 mm. of water, 75 mg. of protein per 100 cc., and 7 lymphocytes and 2 red blood cells per cubic millimeter.

Repeated lumbar punctures gave the following results:

Nov. 8: Initial pressure 440 to 460 mm.; fluid bloody
Initial pressure 280 mm; fluid bloody
Nov. 9: Initial pressure 165 mm.; fluid bloody
Initial pressure 480 mm.; fluid bloody
Nov. 10: Initial pressure 180 mm.; fluid bloody

An electroencephalogram revealed abnormal diffuse and focal slow-wave activity, which was probably correlated with diffuse hypertensive vascular changes.

The patient suddenly became worse and died on Nov. 10, 1950.

Necropsy revealed thrombosis of the posterior half of the superior longitudinal sinus, with sparing of the confluent sinus, and incomplete thrombosis of both lateral sinuses, with recanalization in parts. The pathologist estimated these thrombi to be about four months old, the beginning of thrombosis corresponding closely with the onset of symptoms. There was no evidence of a space-filling lesion or other specific neurological disease. A small hemorrhage in the left temporal lobe and a cerebellar infarction, both recent enough to have occurred during his period of hospitalization, were the only additional findings in the central nervous system. The neuropathologist classified this case under the cerebral pseudotumors.

RÉSUMÉ OF CASES

Headache and blurring of vision, either singly or together, were the presenting symptoms in all our patients. Other prominent symptoms were tinnitus, vertigo, and dizziness; but there was no history or evidence of ear disease. In one instance, these symptoms followed an illness described by the patient as "flu."

Papilledema, with or without hemorrhages, measured from 1 to 4 D., and lasted as long as two years in one case. Visual acuity was generally good, but in Case 7 there was no light perception in one eye on admission. In another case vision was 20/70 in each eye without other apparent cause, but it improved within a week to almost 20/30. In one case bilateral sixth-nerve paresis was present.

The visual fields revealed abnormal enlargement of the blind spots in all but Case 3; and a variety of other defects were found, such as unilateral nasal quadrant defect in Case 2, peripheral constriction in Case 4, and constriction with bizarre changing isopters in Case 5.

The remainder of the neurological examination showed nothing remarkable except in Case 4, in which there were weakness and some cerebellar signs on the left side, these symptoms disappearing in a short time.

Lumbar puncture or the ventriculographic procedure revealed abnormal elevation of the cerebrospinal fluid pressure in all cases, the highest reaching 550 mm. of water. A ventriculogram or pneumoencephalogram revealed normal ventricles in five cases, small ventricles in one case, and questionably small ventricles in another. Electroencephalograms and roentgenograms of the skull showed no localizing abnormalities.

It is known that after ventriculographic or pneumoencephalographic examination or repeated lumbar puncture there may be a temporary increase in the number of red and white blood cells, or of only white blood cells, in the cerebrospinal fluid. Thus, in Cases 1 and 5 an increase of the white blood cells in the cerebrospinal fluid occurred after such procedures. In Case 1 the subsequent cell counts were normal, and in Case 5, even though there was no record of follow-up cell counts, the patient's course falls clearly into that of meningeal hydrops. In Cases 4 and 6 there was bloody fluid, and the ratio of white cells to red cells was within the expected range. Follow-up determinations gave values within normal limits in these two cases. The cerebrospinal fluid protein was slightly elevated in three cases; again, in two of these, Cases 2 and 4, the increase was an isolated finding, not present on other determinations. In Case 7 the cerebrospinal fluid was examined for protein only once, and the level was recorded as 75 mg. per 100 cc. The fact that there was some bleeding at the time of the ventriculographic study in Case 7 might explain a certain amount of the increase in cerebrospinal fluid protein. However, even if this elevation were not due to artifactual bleeding, the clinical picture together with the autopsy report in this case leaves little doubt of the diagnosis.

The course in these cases, as in the cases of other reports, was one of gradual improvement. The ophthalmic findings improved as the other symptoms and signs cleared except in Case 7, in which the outcome was fatal.

Interestingly, moderately severe hypertensive vascular disease was known to have existed for some time prior to admission in Case 7; therefore, the question arose as to the part played by the hypertension in producing the signs of increased

intracranial pressure. It was pointed out by the ophthalmologist that there were only mild diffuse arteriolar changes in the fundi, with no irregularities of the lumina or ensheathing, and the hemorrhages present were limited to the peripapillary area. This was interpreted as indicating that an intracranial cause other than the hypertension was responsible for the papilledema. The medical consultant also was of the opinion that the findings in cardiac and renal function studies were not compatible with the presence of malignant hypertension.

COMMENT

Papilledema was the cardinal sign in these cases, and in a large number of cases it may be the only one. However, its significance differs from that in the average case in which papilledema is seen because the over-all prognosis is much better in this entity.

Dandy [3] observed that after subtemporal decompression the decompression site may be tense or soft at various times during the course of the disease, which may last for years. In some of our cases, too, the follow-up lumbar punctures showed weekly fluctuations between normal and abnormal pressure. This variation might be a factor in explaining why these patients tolerate this disease so well for such long periods.

The treatment undertaken in our cases was repeated lumbar punctures in the hospital, and then in the outpatient department, until the cerebrospinal fluid pressure remained continuously low. The efficacy of any treatment is difficult to evaluate because the natural course of this disease is one of spontaneous remission. However, favorable results have been reported in patients who have had subtemporal decompression,[13] removal of a thrombus from the superior longitudinal sinus,[1] or ventricular taps.[5] Some of the patients treated by the last method seem to have had their course changed for the better.

The ophthalmologist can play an important part in the form of treatment to be employed by carefully observing for signs of increased papilledema, decreased visual acuity, and loss of visual field. Such signs, since there is usually little other evidence of damage on which to depend, would be indications for intervention.

Since almost all patients with meningeal hydrops recover eventually, there has been little pathological material for study except in cases of death from other causes. Therefore the case with postmortem examination presented in this paper is of special interest. Examination of the brain in Case 7 revealed extensive old thrombosis of the superior longitudinal and both lateral venous sinuses, the thrombi having undergone some recanalization. The duration of the thrombi as estimated by the pathologist corresponded closely with the duration of symptoms. However, no history could be obtained which might relate the onset of these thrombi to any antecedent factor, as was done in Symonds' [10] cases of otitis media. Although there is other evidence that this clinical picture is found in thrombosis of the dural venous sinuses, there is a postmortem examination reported by Davidoff and Dyke [2] which failed to reveal any definite pathological changes in the brain to account for the meningeal hydrops.

13. Dandy.[3] Davidoff and Dyke.[2]

The same syndrome has also been reported to occur at the time of the menses in patients who have much edema with their periods.[14] Presumably cerebral edema explains the clinical picture in this condition. It has also been noted to occur in the postpartum state and after head injuries.[1]

Thus, it would appear that the clinical entity of meningeal hydrops can be caused by thrombosis of the dural venous sinuses, cerebral edema, and possibly other mechanisms, such as those suggested by McAlpine [7] and Dandy.[3] It is also evident that the circumstances preceding the onset of this syndrome are diverse.

CONCLUSIONS

Meningeal hydrops is a clinical syndrome in which the outstanding signs and symptoms are headache, blurred vision, and papilledema.

Other important ophthalmological signs, which are occasionally present, are extraocular-muscle pareses and visual-field abnormalities. Since there are usually a minimum of nonocular signs, the ophthalmologist is frequently called upon to chart the course of this disease.

Aside from increased pressure, the cerebrospinal fluid usually shows no abnormality, and ventriculography shows normal or small symmetrical ventricles.

The commonest proved etiologic factor is thrombosis of the dural venous sinuses.

In most cases the prognosis is good for ultimate complete recovery.

14. McCullagh, E. P.: Menstrual Edema with Intracranial Hypertension (Pseudo-Tumor Cerebri), Cleveland Clin. Quart. **8**:202-212, 1941.

OCULAR LESIONS IN PATIENTS WITH PORPHYRIA

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EXCRETION of traces of porphyrins in the urine is a normal occurrence in many mammals, an outstanding exception being the American ground squirrel, Sciurus niger, which normally passes very large amounts in the urine. Adult man passes about 0.1 mg. of coproporphyrin daily in the urine and three to four times this amount of a mixture of porphyrins in the feces. In some hepatic and hematological disorders and in certain types of poisoning, particularly with lead, slight to moderate increases in urinary porphyrin occur, seldom, however, sufficient to impart a recognizably abnormal color to the urine. Almost a century ago notice was first taken of dark-red urines which did not contain blood but which showed absorption bands on spectroscopic examination similar to those of hematoporphyrin, first prepared at about that time by chemical treatment of hemoglobin and its nearer derivatives. It was noted that in some cases the condition was chronic and that there was a history of passing dark urine from infancy, while in others the anomaly occurred intermittently in association with episodes of abdominal colic. In the former group the condition was characterized by extensive scarring of the skin of the exposed surfaces, which was the end-result of bullous lesions, occurring most frequently during spring and summer, and was known as congenital porphyria; in the latter the disorder was distinguished as acute porphyria, and since a history of the use of sulfonmethane (sulfonal®), then becoming a widely used sedative, was often given, it was regarded as a manifestation of toxicity. These cases were few and far between and for long remained clinical curiosities; the presence of grossly increased amounts of porphyrin in the excreta was definitely established, and these complaints are now known as the porphyrias.

CHEMICAL CONSTITUTION

Organic chemists, chiefly in the German schools, showed that porphyrins could be derived from hemoglobin and chlorophyll; much later it was found that myoglobin, the pigment of red muscle; cytochrome; catalase, and other respiratory enzymes also contained heme, and thus on appropriate chemical treatment these, too, could give rise to porphyrins. Porphyrin in combination with iron forms heme, which when combined with various proteins constitutes the various animal respiratory pigments, while porphyrin combined with magnesium is an essential constituent of the chlorophylls. During the second and third decades of the 20th century, the porphyrins excreted by patients with porphyria were isolated and

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studied. It was recognized that several porphyrins existed in nature, and many others were artificially synthesized in the laboratory by Hans Fischer and his colleagues. Of these, only a few need be more fully described for present purposes.

1. Protoporphyrin IX is the residue after removal of the iron from heme. It is found in small quantities in normal feces and the shells of some varieties of birds.

2. Hematoporphyrin is believed to occur only as an artificial product by chemical treatment of heme-containing natural pigments. For this reason, the older terms hematoporphyrinuria and hematoporphyria have been replaced by porphyrinuria and porphyria. This artificial product has been marketed under the trade name "photodyn," and its pharmacological properties have been studied. On injection into animals it produces photosensitivity and edema.

3. Coproporphyrin occurs naturally in two isomeric forms, Types I and III. Type III is most closely related to protoporphyrin IX but is not believed to arise in vivo from the degradation of heme pigments to any great extent. Type I cannot originate from this source and is believed to be synthesized in small amounts during normal hemopoiesis but is excreted without further utilization in the animal economy. Production and excretion are increased in the porphyrias.

4. Uroporphyrin is present in considerable amounts in the urine in porphyrias and is a normal pigment in the feathers of some birds. The existence of Isomer I as the predominant porphyrin in the urine in cases of congenital porphyria has been firmly established. Waldenström [1] recovered a uro- type of porphyrin from the urine of patients with acute porphyria which he identified as the Type III isomer, and other workers have isolated a similar porphyrin in their acute cases. More recently, several workers have claimed that this porphyrin is a mixture of uroporphyrin I and another, as yet incompletely identified, porphyrin. Further study of this point is being pursued at present.

Recent work has indicated the presence of small amounts of porphyrins other than the uro- and copro- types in porphyria urines; these are also being further studied.

Because of their characteristic absorption spectra and brilliant fluorescence in ultraviolet light, the qualitative detection of porphyrins is a relatively simple procedure. Recovery from excreta and purification for the purpose of complete identification are elaborate procedures and, as just indicated, present difficulties which are not yet completely resolved. It should be mentioned at this point that the chemical constitutions of the retinal pigments rhodopsin, porphyropsin, and iodopsin have also been worked out. These are complexes of protein and carotenoids of the vitamin A type and are not chemically related in structure to porphyrins.

CLINICAL TYPES

Brief mention has been made of the various clinical manifestations of porphyria. The congenital type remains a rarity, but other types have been encountered rather oftener and have been reported from many parts of the world. Sweden has the doubtful distinction of having provided the greatest number of cases,[1] but the proportion appears to be high in relation to the population in South Africa.[2] A brief summary of the clinical manifestations follows.

1. Waldenström, J.: Studien über Porphyrie, Acta med. scandinav., Supp. 82, 1937.
2. Barnes, H. D.: Further South African Cases of Porphyrinuria, South African J. M. Sc. 2:117, 1951.

Congenital Porphyria.—The diagnosis is made on the basis of pronounced porphyrinuria, which is chronic and, on inquiry, can be traced back to early infancy. The anomaly probably exists before birth, since the milk teeth are deeply stained with porphyrin. The bones are subsequently found to be deeply pigmented, resembling mahogany in color. The skin is subject to bullous eruptions, mostly confined to the exposed parts, and in countries where the sunshine varies markedly with the seasons these lesions first appear, and are most prevalent during the sunny periods. Trauma appears to play a part in the etiology, so that the lesions are variously known as hydroa vacciniforme seu aestivale and epidermolysis bullosa. Subsidence of the bullae is followed by a protracted stage of crustation, and healing leaves scars, sometimes fibrous and sometimes representing a tissue-paper atrophy. Pigmentary changes are common, and hirsutism is a feature seen mostly in females. Marked deformity and mutilation are characteristic of long-standing cases; splenomegaly, hepatomegaly, and a degree of anemia are also late events. The psychotic, neurological, and abdominal symptoms of acute porphyria do not occur in this type. The disease has been reported in cattle and swine.

Delayed Cutaneous Porphyria.—The picture in this condition is almost identical with that in the preceding type, but onset is deferred until the end of the second decade or later, and the patients are very positive about their freedom from symptoms earlier in life. The degree of porphyrinuria is usually much less marked; indeed, the urine may suggest in appearance the presence of a slight excess of urobilin, or may even appear normal, but spectroscopic examination of a deep layer shows the presence of characteristic bands. The skin lesions vary from facial hyperpigmentation with dermatitis, easily confused with pellagra, to extensive bullosis with scarring (Fig. 1*A-D*), but deformity and mutilation have not yet been seen in cases of this type. Many patients give a history of alcoholism, and there is often evidence of disturbed liver function.

Acute Porphyria.—This form is characterized by episodes of pronounced porphyrinuria, accompanied by vomiting, severe abdominal colic, extreme constipation, peripheral neuritis, sometimes progressing to flaccid paralysis of one or more limbs, and various personality changes, though these symptoms are not necessarily all present together and may vary from one episode to another. The attacks may recur within a few weeks or be separated by many years. In some cases water and electrolyte imbalance occurs, not due to vomiting only, since the urine may continue to contain chlorides even when the plasma chloride is greatly reduced. This type of porphyria is characterized by the presence in the fresh urine of porphobilinogen, a colorless substance which soon reverts to a deep-red pigment, known as porphobilin. It is this change which accounts for the marked darkening of the urine on standing, especially when acidified and exposed to light. This substance is not present in the urine in the types of porphyria previously described. It has, however, been found occasionally, together with porphyrin, in the urine of patients with the latent form, who have always been the relatives of patients with acute porphyria.

Comment.—While the distinction between the delayed and the acute type of porphyria is fairly clear in the majority of cases, instances have been reported in

which porphyrinuria was associated with some of the features of both types. Moreover, close inspection will sometimes show evidence of past dermatological abnormalities in what appears to be a case of frank acute porphyria, while in other cases the disease begins as delayed cutaneous porphyria and later reverts to the acute form with no active skin lesions.

There is evidence of a familial incidence in all these forms of porphyria, thus suggesting a genetic basis for the complaint. Experience to date of a larger number of cases in South Africa than the 84 to be described has been that the acute form

Fig. 1 (Case 66).—Photographs of non-European woman in case of delayed cutaneous porphyria: (*A*) temporal hirsutism and pigmentary changes on site of old lesion on forehead. Note blepharochalasis. (*B*) Pigmentary changes on site of old lesion on medial aspect of right foot. (*C*) Depigmented scars on hand with hyperkeratotic thumb nail. (*D*) Bulla on middle toe of left foot and hyperkeratosis of nail of big toe.

is very much commoner in persons of European ancestry, while the type with dermatological manifestations is predominant among non-European subjects.

The integrity or otherwise of the oculomotor function is often reported in the histories of patients with porphyria, and ophthalmological lesions of the external structures have been described by several authors; but examination of the fundus, according to the literature available, has received scant attention. Except for occasional transient amaurosis, the patients rarely complain of visual disturbances, a fact which may be explained by the peripheral distribution of most of the fundus lesions. Very little information is to be obtained from the textbooks.

PRESENT STUDY

Our case material comprises 38 subjects of European ancestry and 46 non-Europeans; 50 were females and 34 males; their ages ranged from 8 to 62 years.

Ocular Adnexa.—The eyebrows may be thinned or thickened, the latter forming part of a degree of facial hirsutism in which the eyebrows meet in the midline, but more commonly the outer extremities extend upward to meet a temporal extension of the scalp hair (Fig. 1*A*). Oculomotor paralysis was not encountered in this series. Scarring of the lids and pigmentation following the characteristic bullous lesions may be found, and in severe cases result in gross deformities and ectropion.[3] Edema of the lids may occur when there is an eruption of the skin in neighboring sites. Photophobia and blepharospasm, when present, are usually associated with more widespread sensitivity to light. Loss of elasticity of skin, where scars do not determine otherwise, is often seen. Laxity of skin from atrophy gives equal or unequal blepharochalasis on the two sides early in life—in some

TABLE 1.—*Clinical Findings in Conjunctiva and Eyelids in Eighty-Four Cases of Porphyria, Classified as to Race, Sex, and Age*

Name of Condition	No. of Affected Patients	Males*		Females*		Age Groups*					
		Europeans	Non-Europeans	Europeans	Non-Europeans	1-10	10-20	20-30	30-40	40-50	50-60
External scarring of lids	9	7	53	13; 14; 19; 29	56; 5; 37	..	5	13; 37	29; 56	7; 14; 19; 33	..
Internal scarring of lids	3	..	25	9	28	..	..	9; 25	28	..	..
Ectropion	3	7	33	..	56	..	..	..	56	7; 33	..
Blepharochalasis	11	55; 68	..	18; 19; 29; 57; 63; 84	32; 66; 70	..	18	55	29; 32; 57; 63; 66; 84	19	70; 68
Symblepharon	4	8	33	9; 19	..	..	..	9	8; 33	19	..
Hyaloid thickening of conjunctiva	6	3	..	63	4; 28; 37; 75	..	3	37; 75	63; 4; 28	..	..
Pemphigoid occlusion of fornix	1	..	..	..	32	..	..	32	..	..	..
Acute bullous conjunctivitis	2	..	..	42; 51	..	..	..	..	42	..	51

* In this table, and in the accompanying tables, the serial numbers of the patients are recorded.

cases at the age of 20. It may be accompanied with abnormal creasing of the skin. This is mostly found in the delayed cutaneous porphyrias, but has also been seen in a case of acute porphyria. The lids were affected in 26 of the 84 patients examined (Table 1).

Conjunctiva.—The conjunctiva being an easily accessible part for examination in the ward, the lesions in this site have been the most frequently described of the ocular conditions in porphyria. The conjunctiva was affected in 13 of the 84 patients, exclusive of the 3 whose conjunctiva was exposed in ectropion (Table 1). Congestion and reactivity appear without bullae being present anywhere in the neighborhood of the eyes, although occurring elsewhere on the body. Blisters forming on the conjunctiva (Fig. 2) may result in symblepharon of varying degree, sometimes so severe that the entire conjunctival sac is shrunken, resembling

3. (*a*) Sorsby, A.: Genetics in Ophthalmology, London, Butterworth & Co., Ltd., 1951, p. 203. (*b*) Lichtwitz, L., in Schieck, F., and Brückner, A., Editors: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1932, Vol. 7, p. 30. (*c*) Sorsby, A.: Systemic Ophthalmology, London, Butterworth & Co., Ltd., 1951, p. 68.

the end-result of pemphigus.[4] Conjunctival bullae, although they look like blisters, contain no large fluid spaces, and the antecedent lesion resembles phlyctenular conjunctivitis in a perilimbal position.[4b] The palpebral conjunctiva shows scars under the upper, but oftener under the lower, lid, similar to the star-shaped scars of trachoma, but oftenest resembling the *en plaque* type of trachoma scar. Some cases of vernal conjunctivitis (spring catarrh) show porphyrin in abnormal quantities in the urine.[5] We have not yet investigated this aspect of porphyria. A condition which often arrests the attention of observers is the presence of pigment in the interpalpebral fissure of the Bantu,[6] but this is very commonly seen in Bantu subjects and cannot be associated with porphyria, in the light of our present experience. Marked thickening of the conjunctiva, following more or less the interpalpebral area and appearing very similar to the thickening of hyaloid conjunctival degeneration,[7] may be seen in many of these cases.

Sclera.—The lesion oftenest seen on the sclera is referred to as scleromalacia perforans [8] (Fig. 3). This lesion appears in both the congenital and the delayed

Fig. 2.—Photograph of conjunctival "bulla" seen in delayed cutaneous type of porphyria. These are actually lesions of severe conjunctival edema.

cutaneous type of porphyria. The lesions may appear on the sclera only or may be accompanied with severe "keratomalacic ulcers" and corneal scarring (Fig. 4). One author [8a] claimed that administration of riboflavin (vitamin B_2) and folic acid had a beneficial effect on such lesions. We consider that this lesion as seen in cases of porphyria differs from true scleromalacia perforans. Penetration is

4. (*a*) Lichtwitz.[8b] (*b*) Cohen, M., and Sulzberger, M. B.: Essential Shrinkage of Conjunctiva in a Case of Probable Epidermolysis Bullosa Dystrophica, Arch. Ophth. **13**:374, 1935.

5. (*a*) Langecker, H.: Zur Fluoreszenzbeobachtung bei den Porphyrien, Ztschr. physiol. Chem. **115**:1, 1921. (*b*) Junius, P.: Der Frühjahrskatarrh, Arch. Augenh. **87**:1, 1920. (*c*) Langecker, H.: Frühjahrskatarrh und Porphyrin, ibid. **89**:118, 1921.

6. Findlay, G. H., and Barnes, H. D.: Congenital Porphyria, Hydroa Aestivale and Hypertrichosis in a South African Bantu, Lancet **2**:846, 1950.

7. Kloppers, P.: Demonstrasie van geval van kroniese porphyrinurie, Christo Beyers Medical Society, Oct. 7, 1941, unpublished work.

8. (*a*) Kadlecova, V.: Xeroftalmie a keratomalacie u idiopaticke kozni porfyrie, Československ. oftal. **4**:154, 1948; abstracted, Excerpta med. Sect. XII **3**:302, 1949. (*b*) Zavalia, U.; Allende, I. M., and Oliva, R. O.: Scleromalacia During Chronic Porphyrinuria, Arch. oftal. Buenos Aires **12**:115, 1937.

more rapid and is seldom complete, but when perforation occurs it does so in one attack; after healing a subsequent lesion seems not to occur at the same site. Furthermore, the lesion is not restricted to the sclera but spreads over the limbus and encroaches on the cornea, remaining of equal depth on the two anatomical sites (Fig. 4). The conjuctiva which re-forms over the lesions where open ulceration was present shows linear streaks of scar tissue. How far the sclera

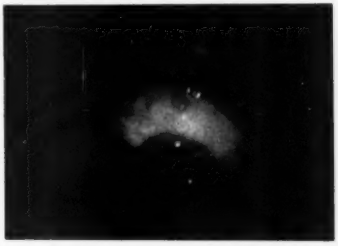

Fig. 3.—Photograph of keratomalacia perforans. Small round staphyloma can be seen on sclera opposite 12 o'clock on limbus.

Fig. 4.—Photograph of scleromalacia where lesion extends onto cornea. Tiny arrows indicate borders of lesion. Cornea is scarred from earlier ulceration.

of the posterior segment can become affected we do not know, but we have yet to see a lesion on the sclera posterior to the level of insertion of the rectus muscles. Probably the acutely reactive stage with loss of the overlying conjunctiva has been described as porphyric scleral ulcers [5b] (Table 2).

Cornea.—The cornea may present a keratomalacic type of lesion,[5a] e. g., perforation and scarring,[3] as well as leucoma adherens.[5b] Because the keratomalacia,

as it is usually called, is usually severe and acute, the corneal scars are dense, and blindness results (Fig. 4). Since the leucoma adherens of porphyria usually touches or overlaps the limbal margin, it is difficult to decide whether the origin is corneal or scleral. In the Bantu the dense leucoma has pigment patches on it, but no associated fluorescent spots have been found on microscopic examination in ultraviolet light. Corneal participation in light sensitivity has been referred to by several workers,[3] but in this series of cases was no greater than one usually finds in keratitis. Lesions resembling groups of phlyctenules may precede actual corneal ulceration. One of our patients with a severe ectropion of the upper and lower lids had exposure keratitis. This was regarded as purely secondary.

Iris and Lens.—There is little to be said about the iris except that it participates in leucoma adherens. A slight sifting of iris pigment into the lower iridocorneal angle was observed in a Bantu patient of 33, and in another, of 13 years with congenital porphyria. A European woman aged 34 showing complete degeneration of the left fundus had a partly disintegrated cataract with ruptured and rolled-back

TABLE 2.—*Scleral and Corneal Conditions Found in Eighty-Four Patients with Porphyria, Classified as to Race, Age, and Sex*

Name of Condition	No. of Affected Patients	Males*		Females*		Age Groups*					
		Europeans	Non-Europeans	Europeans	Non-Europeans	1-10	10-20	20-30	30-40	40-50	50-60
Scleromalacia perforans	2	..	..	19	37	..	..	37	..	19	..
Scleromalacia semiperforans	5	..	33; 79	51	5; 37	..	5	37	79	33	51
Extension on to cornea	3	..	33	..	5; 37	..	5	37	..	33	..
Limbal phlyctenosis, later developed farther	2	..	25	..	32	..	..	25; 32	..	..	..
Leucoma adherens	5	..	33; 59	42	5; 32	..	5	32; 59	42	33	..
Keratitic scars	5	..	6; 25	18	32; 49	..	18	25; 32	49	6	..

* Serial number of each patient is recorded.

anterior capsule in the right eye and slight haziness of the posterior cortical layers of the left lens. During the acute and postacute stages of moderate and severe lesions of the fundus we observed sluggish reactions of the pupil. Jaffe [9] recorded unresponsive pupils. During attacks of acute porphyria, two of our patients had very painful eyeballs, for no apparent reason, and transitory myopia.

Ocular Fundus.—The ocular fundus has occasionally been referred to in clinical and experimental [10] studies on porphyria. Pallor of the optic nerve head has been ascribed to secondary anemia after hepatic damage.[11] Blurred disks,[9] retrobulbar neuritis,[12] and doubtful optic neuritis before death [13] have been briefly mentioned. We have seen a pale disk, a secondary phenomenon, in a case with very extensive old chorioretinitis, but in two cases of acute porphyria during attacks the disks

9. Jaffe, N. S.: Acute Porphyria, Am. J. Ophth. **33**:470, 1950.

10. Sai, E.: Haematoporphyrin: Action of Displacement of Retinal Pigments, Folia pharmacol. japon. **31**:1158, 1940.

11. Walsh, F. B.: Clinical Neuro-Ophthalmology, Baltimore, Williams & Wilkins Company, 1947, p. 849.

12. Ford, F. R.: Diseases of the Nervous System in Infancy, Childhood and Adolescence, Ed. 2, Springfield, Ill., Charles C Thomas, Publisher, 1944, p. 709.

13. Stockton, R.: Tr. A. Am. Physicians **15**:6, 1900; cited by Ford.[12]

were slightly blurred and hyperemic. The only detailed description of fundus lesions during an attack of acute porphyria comes from Jaffe, who described them as unusual. Jaffe [9] stated:

> Ophthalmic examination revealed bilateral ptosis of the eyelids. Levator action was negligible on both sides. There was bilateral, incomplete oculomotor nerve palsy. The pupils were dilated and immobile to light or near stimuli. Both eyes could be abducted well. On attempted downward gaze, the eyes intorted, indicating good function of the superior oblique muscles. Convergence was impossible. Examination of the optic fundi revealed bilateral blurred discs and

TABLE 3.—*Clinical Findings in Fundi of Eighty-Two Patients with Porphyria Classified as to Race, Sex, and Age*

Name of Condition	No. of Affected Patients	Males*		Females*		Age Groups*					
		Europeans	Non-Europeans	Europeans	Non-Europeans	1-10	10-20	20-30	30-40	40-50	50-60
Group 1											
Subgroup A											
Cotton-wool patches of edema	3	..	44	10	34	..	10; 44	34	..	..	..
Above associated with hemorrhage	1	..	..	10	..	..	10	..	..	..	..
Subgroup B											
Flat striate edema	4	..	58	11; 17; 19	..	..	..	17	58	11; 19	..
Group 2											
Subgroup A											
Discrete annular choroiditic types	10	55	45	29; 42; 63; 84	4; 28; 37; 70	..	45	37; 55	4; 28; 29; 42; 63; 84	..	70
Subgroup B											
Large en plaque type	6	8	..	9; 13; 18; 48	70	..	18	48; 9; 13	8	..	70
Above with edema and hemorrhage	1	8	..	..	..	..	..	..	8	..	..
Group 3											
Lanceolate and striate scars	13	8	25; 38; 59	1; 17; 19; 18; 14; 21; 84	43; 70	38	18; 21; 43	17; 25; 59	8; 84	19; 1; 14	70
Granular diffuse lesions	10	16	53; 2; 38; 59	27	28; 43; 56; 70	16; 38	27; 43	2; 59	53; 28; 56	..	70
Macular pigment shift	12	16	..	48; 14; 21	70; 71; 73; 79; 5; 43; 82; 83	16	5; 21; 43	48; 73	71; 83	14; 79	70; 82
Globular pigment scars	9	8	44	9; 13; 15; 18; 21	56; 70	..	44; 18; 21	9; 13; 15	8; 56	..	70
Depigmented patches	5	..	..	84	28; 65; 70; 76	..	..	..	28; 84	65; 76	70
Hemorrhages present	3	8	53	..	43	..	43	..	53; 8	..	..

* Serial numbers of patients are recorded.

> numerous hemorrhages which were nerve fiber, preretinal, and choroidal in distribution. Most of the hemorrhages were in the vicinity of the optic discs, the maculas, however, being spared. There was no retinal pallor or edema. The vessels were of normal caliber. There were no evidences of hypertensive retinopathy. [By the tenth day] most of the flame-shaped hemorrhages were gone and the others were absorbing readily.

These findings to some extent correspond with our experience in acute cases. The lesions encountered can best be described under three main groups according to their appearances (Table 3). Examination of the fundus was impossible in two cases with dense corneal opacities.

1. The lesions which are seen mostly during cerebral episodes of acute porphyria are situated mainly in the retina and are edematous in their early stages. They are of two kinds:

(*a*) A type which in the acute stage resembles white patches of edema ½ to 1 disk diameter in size, looking like cotton-wool, round, and somewhat fluffy (Fig. 5*A*). These may last from one to four weeks. When of short duration, 7 to 10 days, these patches leave no detectable scar, except occasionally a diffuse, slightly pigmented area. In two cases the end-results of the longer-lasting lesions were rounded blobs of very black pigment (Fig. 8, quadrant II), not unlike melanoma, but blacker than this tumor. They show a tendency to form near blood vessels, but the macular area is avoided. In one instance a few deep petechial hemorrhages were seen near and inferior to the optic disk.

(*b*) A type showing no predilection for the site of its appearance in the fundus, most such lesions having been observed in the periphery. They appear to be thin flat edematous films, which are usually slightly striated, like a peripheral myelin-sheathed patch of retinal fibers, and, like it, the striations are more marked at the

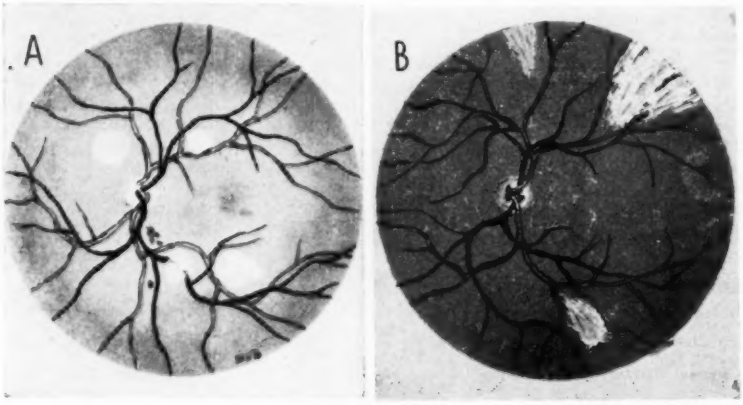

Fig. 5.—*A* (Case 34), drawing of round, cotton-wool-like patches of retinal edema seen during attack of acute porphyria. Petechial and interfibrillary hemorrhage are shown.

B (Case 53), drawing of fundus depicting flat, slightly striated type of retinal edema seen during acute attacks.

peripheral and the central borders (Fig. 5*B*). The broader base of the patch is turned away from the disk. They may also appear as very thin, nebulous plaques of edema, and their localization seems to be in the retinal fiber layer. In all cases of lesions of this type there are a general paleness of the choroid and a hazy, slightly swollen disk, both phenomena persisting any time up to a month after the acute attack of porphyria, while the patches of edema usually disappear within three weeks after the attack is over. Subsidence of the lesion leaves a diffusely granular patch of chorioretinal pigment over the same area, but after the severer and more protracted lesions a striated pigment scar forms (Fig. 8, quadrant IV).

The round cotton-wool patches and the flat striated films of edema have not yet been seen together, and in none of the cases of the latter type of edema were there associated hemorrhagic phenomena. Both these types of edema occur with the cerebral type of acute porphyria.

2. The commonest choroidal lesion seen in the congenital and delayed cutaneous forms of porphyria is the scarred stage. However, this has been seen in various stages and can also be divided into two subgroups.

(*a*) The slowly progressive, discrete annular type. The early annular lesion is but slightly pigmented, having a limiting border of increased pigment (Fig. 6*A*). The typical lesion has an outer slightly pigmented border, inside which there is a pale-yellow choroidal zone (Fig. 6*A*). Inside this, again, is the body of the lesion, which is a heavily pigmented, broader, and more irregular zone, consisting of granules of black pigment of varying shades. In non-Europeans and in brunet Europeans this ring encloses a less heavily pigmented area (Fig. 6*B*), which in blond patients is usually white, denuded sclera, occasionally containing discrete granules of brown pigment (Fig. 6*C*). In patients who have had porphyric symptoms for a long period these discrete annular lesions cover the whole fundus, leaving the macular area relatively free but slightly scarred, with an irregularly pigmented degeneration (Fig. 6*B* and *C*). Lesions of this group do not increase in size; they mature slowly, and occasionally the overlying retina shows slight edema and later becomes involved, as evidenced by pigment sheathing of the vessels. While it appears that these lesions generally remain discrete, the irregular distribution of pigment in and about some of them suggests coalescence. This may be explained by the formation of new lesions sufficiently near the older ones for overlapping to occur. Sight is sometimes severely impaired.

(*b*) Diffuse, rather dense choroiditic lesions which spread out from the disk. The dominant impression is that of an *en plaque* lesion, reminiscent of a clover leaf in shape (Fig. 6*D* and *E*). It may extend as much as 4 diameters from the disk and cover the macula. The lesions are uniformly pigmented, but white bands of fibrous tissue may be seen inside the long linear "pseudopodia," which extend toward the periphery. The retinal vessels are not enveloped by pigment. In addition, one may observe in the periphery clusters of three to five round, black "globules," similar to those which arise from the cotton-wool type of edema described above (Fig. 6*D*). The periphery also shows conspicuous patchy granularity (Fig. 6*E*), although the fundus between this and the central lesion remains singularly clear. The involvement is unilateral, and patients describe the onset of blindness in the affected eye as starting with the seeing of black spots and progressing to gradual loss of vision over a day or two. The slight visual improvement that usually occurs some months after the known types of central retinal hemorrhage does not follow. We examined a patient with such a lesion two weeks after the onset of blindness and saw flat, large, deep-lying hemorrhagic areas with overlying patches of retinal edema. The appearance was that of a large choroidal hemorrhage surrounding the optic disk, which was slightly swollen and contained no blood in its tissues.

3. In a miscellaneous group are placed the doubtful choroidal lesions. The high incidence of these lesions in cases of porphyria and their frequent presence in young subjects merit their being recorded, though at this stage they are not regarded as typical. Another form fairly frequently observed consists of a diffuse shift of macular and paramacular pigment to form patches of irregular pigmentation, often

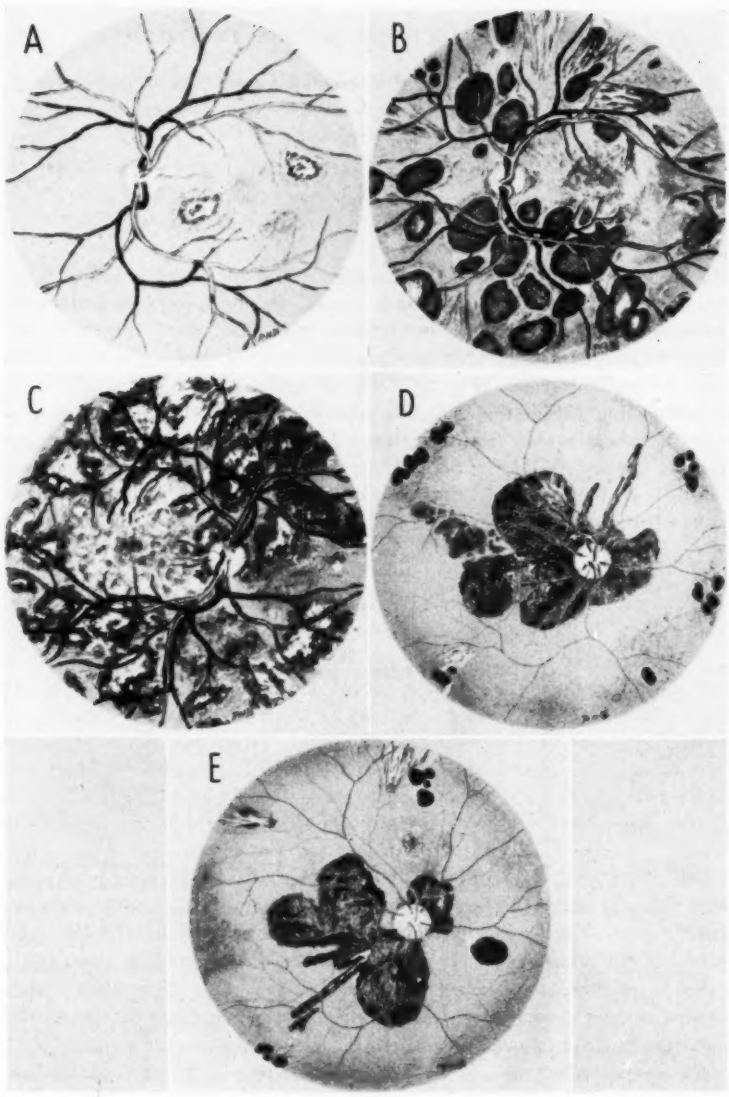

Fig. 6.—*A* (Case 28), drawing of fundus showing discrete annular type of choroiditic lesion in unusual position of proximity to macula. Lower right-hand twin lesion depicts early stage of development; these lesions usually mature without enlarging or fusing. Upper two, large lesions are fully developed, demonstrating concentric rings of pigment and atrophy.

B (Case 29), drawing of fundus of darkly pigmented European woman. In upper part of field is discrete annular, heavily pigmented type of choroiditic lesion with patches of striate type.

C (Case 55), fundus drawing, showing extensive discrete annular type of choroiditis with diffuse pigmentary type of macular degeneration. Uppermost, large lesion appears to be fusion of two discrete ones.

D (Case 8), fundus drawing, showing *en plaque* lesion of peripapillary choroiditis and preponderance of clusters of black pigment discs, or "globules," in periphery. A linear pseudopodium points toward 2 o'clock.

E (Case 9), fundus drawing, showing large patch of peripapillary choroiditis, above which is rounded, partially nonpigmented patch, striated peripheral scar, at 12 o'clock, and area of diffuse granularity peripherally in region of 9 o'clock.

showing edematous spots (Fig. 7). A third form is a lanceolate patch of partially denuded sclera pointing centripetally (Fig. 8, quadrant I), which may occur alone or associated with a lesion consisting of diffuse aggregations of thin pigment on a paler choroidal background (Fig. 8, quadrant III). The latter could have underlain a patch of retinal edema at an earlier date. One frequently finds intensely dark "globules," or blobs, mainly in the periphery of the fundus, singly (Fig. 8, quadrant II) or in clusters (Fig. 6*D*). We have seen them result from patches of edema, but this is probably not their only origin, for they have been observed in a developing stage along with deep hemorrhages similar in shape and distribution. This

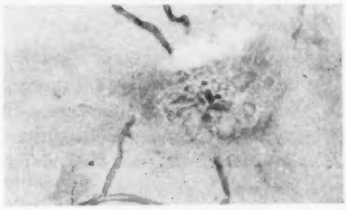

Fig. 7 (Case 71).—Drawing of macular area of patient aged 36, showing shifting of macular pigment in degenerative lesion and two patches of edema below lesion. Visual acuity, 6/18.

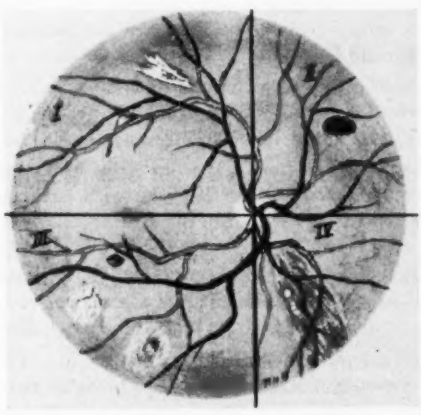

Fig. 8.—Composite drawing of some of lesions of Group III (miscellaneous). Quadrant I, lanceolate scar; Quadrant II, single lesion of "globular" type, resembling nevus or melanoma of choroid; Quadrant III, two lesions of partially nonpigmented type and one dense black "globular" lesion; Quadrant IV, striated patch of edema.

miscellaneous group of lesions can be seen in association with each other and with the foregoing retinal and choroidal types (Fig. 6*B*, *D* and *E*).

When a fundus lesion is discovered which conforms to one of those described in this paper, it is worth while to investigate the case for other signs of porphyria. Examination of the urine clinches the diagnosis if carried out when porphyrins are being excreted, and the history may provide other presumptive evidence. We have not been able to obtain material for histopathological study from any of these cases.

COMMENT

Nineteen of 38 Europeans and 11 of 46 non-European patients examined showed no ocular lesion of any kind. Thirty-one of the 84 patients presented an external lesion. Only 82 could be examined ophthalmoscopically, of whom 42 showed lesions of the fundus. Over several years, one of us had had referred to him 7,548 patients, men, women, and children of all ages, who are members of medical benefit societies in the mining area of the Witwatersrand. Records show that only 311 of these patients had fundus lesions of any kind. Even allowing for the fact that benefit society patients are frequently referred to a consultant for trivial reasons, this proportion is strikingly different from the 42 patients with fundus lesions found in 82 patients in the present series.

The changes recorded in the external eye correspond in the main with earlier descriptions and need no comment. Interest was focused primarily on the internal eye, as little about this part was found in the literature. The record here presented is mainly descriptive, and work is continuing on additional cases. The lesions in the fundus have been seen in various stages of development, but old-established lesions have been seen most frequently.

1. The round, cotton-wool patch type of edema often results in the formation of dense black "globular" patches of pigmentation when the edema has persisted for about three weeks. Edema of shorter duration leaves a diffuse granularity on subsidence, and sometimes no residue can be seen. The lesions tend to persist longer when they are situated in the periphery of the fundus; hence this region should be examined for old lesions.

2. The flat, thin patches of edema leave a diffuse granularity over the affected area, or they may leave thin scars which have a yellowish depigmented or denuded scleral background with pigment striations. They sometimes leave no trace whatever.

3. Hemorrhages are of three kinds. The preretinal hemorrhage leaves no trace on absorption. The interfibrillary hemorrhage leaves a lanceolate or striated scar or a diffusely pigmented patch. The deep massive or deep petechial hemorrhage may be localized in the external granular layer of the retina or in the choroid; these leave dense pigment plaques or "globules," respectively, and there may even be proliferated scar tissue present in the final *en plaque* type of lesion.

4. The discrete annular type of choroiditic lesion is dependent in its depth of pigment on the natural pigmentation of the patient's fundus. These lesions develop and progress slowly over about a five-month period. Small patches of edema may sometimes be seen over a fresh lesion. The lesions remain the same size throughout their development toward the full degree of pigmentation, which is apparently not related to the occurrence of systemic attacks of porphyria.

SUMMARY

Brief outlines of the chemical structure of porphyrin pigments and the clinical manifestations of porphyria are given, followed by a description of the ophthalmological lesions found in 84 cases of porphyria. The incidence of several lesions of the fundus is high and merits more study than has been accorded it in cases so far published.

Drs. Findlay, Grotepas, and Perold, of Pretoria Hospital, permitted the study of their cases.

SIMULATED DIVERGENCE PARALYSIS

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SOUTH HAVEN, MICH.

The manifestations of a typical case of divergence paralysis include the following:

1. Homonymous diplopia, due to an abnormal convergence position, arises rather suddenly. It occurs for all objects beyond a certain distance, mostly beyond 10 to 20 inches.

2. The angle of squint is small or moderate, and does not increase when the patient looks to the right or left; it will either remain the same as in the primary position of the eyes, or it will even decrease.

3. When an object is brought nearer to the patient, the two images approach each other and are finally fused when the object is at a disance of from 10 to 15 inches. At this distance binocular single vision is maintained when looking to the right as well as to the left.

4. When the object is brought still nearer, insufficient convergence causes crossed diplopia.

5. Appropriate prisms, base out, give the patient binocular single vision, even of distant objects in the whole field of fixation.

6. In repeated examinations at different times the angle of squint is found to be relatively constant.[1]

BIELSCHOWSKY, in discussing further these findings in divergence paralysis, stated[2]:

> I have observed many patients with paralysis of one or both abducens nerves in whom typical symptoms were present at first, but the characteristic symptoms were gradually lost and a concomitant type of deviation developed so that it no longer increased, or, on the other hand, decreased, when looking to the right and left. Sometimes this transformation may even occur within a few days. If such a paralysis is seen only in the later atypical stage, it is difficult to distinguish it from a divergence paralysis.

At the Infirmary motility clinic it has been our good fortune to see a number of these patients with paralysis of the lateral rectus muscles in whom, after a period, symptoms developed that were difficult to differentiate from true divergence paralysis.

A study of all our cases in which the paralysis simulated divergence paralysis revealed (1) that in most of them the condition resulted from paresis of one or both lateral rectus muscles, congenital, acquired, or postsurgical; (2) that in some

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1. Bielschowsky, A.: Lectures on Motor Anomalies, Hanover, N. H., Dartmouth College Publications, 1940, p. 104.

2. Bielschowsky,[1] p. 105.

it resulted from accommodative excess for distance only, and (3) that most of them seemed to fall into the classification Group II, esotropia with bilateral depression in adduction.[3]

Since it was believed that these cases were of more than passing interest, I should like, in this paper, (1) to present some of them in detail and (2) to offer a possible explanation for their occurrence.

PRESENTATION OF CASES

In the following three cases of simulated divergence paralysis, the cause was a clinically observed acquired lateral rectus paralysis.

CASE 1.—M. J. M., aged 79, came to the Infirmary in October, 1949, with a history of double vision for distance of six months' duration and no diplopia for near. She had hypertension and thrombophlebitis in the legs.

Examination in October, 1949, showed 15 degrees of esotropia for distance, straight eyes for near, and limitation of movement of the left lateral rectus. A diagnosis of paralysis of the left lateral rectus was made.

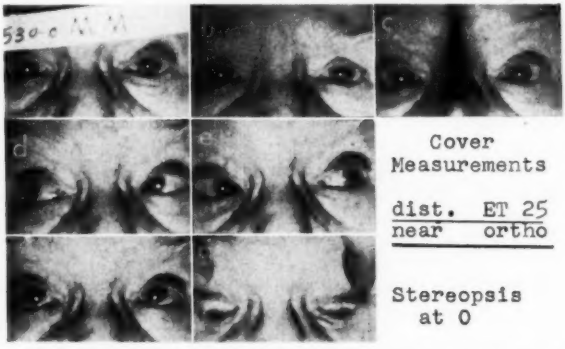


Fig. 1.—(*a*) Right esotropia of 10 degrees for distance with homonymous diplopia (seen clinically, not shown well with photographs); (*b*) no deviation for near, stereopsis at zero; (*c*) near point of convergence 60 to 80 mm.; (*d*) eyes level on gaze to right (slight depression in adduction seen clinically), no limitation of right eye in abduction; (*e*) slight depression of right eye in adduction, no limitation of left eye in abduction; (*f*) 15 degrees of left esotropia on looking up, homonymous diplopia; (*g*) no deviation on looking down and no diplopia.

The findings in May, 1950, 14 months after onset of symptoms, are shown in Figure 1. There was an esotropia of about 10 degrees for distance and of 15 degrees on looking up. The eyes were straight for near and on looking down. There was slight bilateral depression in adduction, which was difficult to photograph. The near point of convergence was 60 mm. Homonymous diplopia was present for distance and did not change perceptibly on gaze to the left or to the right. A red glass placed over the left eye revealed slight left hypertropia on gaze to the left;

3. The characteristics of cases of esotropia with bilateral depression in adduction, viz., (*a*) that the esotropia is greater for distance and upward gaze than for near and downward gaze; (*b*) that right hypertropia is found on gaze to the right and left hypertropia on gaze to the left; (*c*) that the near point of convergence can be good or remote, and (*d*) that the delayed cover test usually shows some type of dissociated vertical divergence, are discussed in a previous paper (Urist, M. J.: Horizontal Squint with Secondary Vertical Deviations, A. M. A. Arch. Ophth. **46**:245 [Sept.] 1951).

a red glass placed over the right eye revealed slight right hypertropia on gaze to the right. Third-grade fusion was present for near with no diplopia. The delayed cover test showed alternating hypophoria.

An 8 △ prism base out over each eye completely relieved the diplopia for distance. However, the patient would not wear the prisms, as she claimed the diplopia did not trouble her much and the prisms were inconvenient and too heavy to wear.

The cause of the esotropia in this case was acute paralysis of the left lateral rectus, very likely on a vascular basis. After one year the limitation of the left lateral rectus had completely disappeared. The residual findings of the remaining weakness of the left lateral rectus were an esotropia for distance and upward gaze and diplopia in these positions. The diplopia findings were similar to those of divergence paralysis. The straight eyes for near and downward gaze, the stereopsis, and the absence of diplopia, along with the convergence near point of 60 mm., indicated that no overaction of the left medial rectus was present. If overaction of the medial rectus had been present, the eyes would have been crossed for near and downward gaze, and there would have been a very close convergence near point.

This case demonstrated that paralysis of a lateral rectus which has become concomitant could have such a minimal effect on the positions for near and downward gaze that the eyes could be straight in these positions. Most likely the eyes were kept straight by the ability of the fusion faculty to inhibit convergence and thus prevent overaction of the medial rectus muscles in the interest of maintaining single binocular vision.

CASE 2.—D. H., aged 21, came to the Infirmary on May 2, 1946, with a history of acute onset of double vision and crossed eyes, of four weeks' duration.

Examination revealed 25 degrees of alternating esotropia, homonymous diplopia in all fields of gaze, bilateral weakness in elevation of the eyes up and in, and bilateral limitation of the eyes in abduction. An admitting-room diagnosis of bilateral paralyses of the lateral rectus and inferior oblique muscles was made. The patient was advised to enter the hospital for a complete work-up but did not return to the clinic until January, 1950, four years later.

During those four years the eyes had been crossed, and there was constant diplopia for distance, while the eyes could be straight for near and there was no diplopia when reading. An admitting-room diagnosis of old divergence paralysis was made at this time.

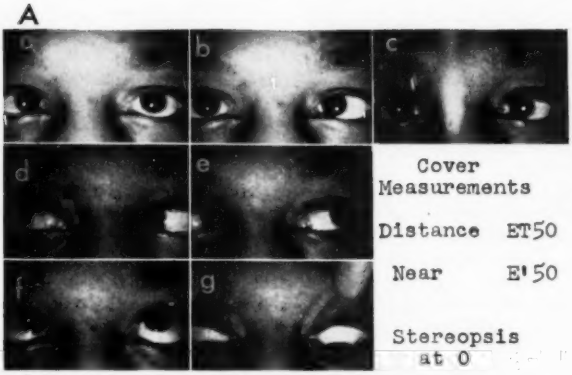
The findings in February, 1950, are shown in Figure 2*A*. There was a constant esotropia of 25 degrees for distance and upward gaze with homonymous diplopia for distance, which was the same on gaze to the right or to the left. The two images were fused 0.33+ meter from the eyes. For near, there was a heterophoria part of the time. The eyes were straight on looking down, with stereopsis in the reading position. The bilateral depression in adduction was pronounced. Unfortunately, measurements with the cover test were not obtained in all positions of gaze.

With homatropine cycloplegia, refraction was as follows:

O.D.: + 1.00 sph. = 20/20
O.S.: + 1.00 sph. = 20/20

Operation, on May 10, consisted of bilateral 8-mm. resection of the lateral rectus muscle. Postoperative findings on Sept. 28, 1950, are shown in Figure 2*B*. The eyes became straight for distance and upward gaze with stereopsis. There was no diplopia in any position of gaze. The depression in adduction was much improved.

The esotropia was caused by the acute onset of gross bilateral paralysis of the lateral rectus, of unknown origin. After four years of constant dissociation for distance, the bilateral paralysis of the lateral rectus improved to such an extent that no limitation was apparent on lateral rotations, and the findings were similar to those of divergence paralysis. The esotropia which was present for distance and upward gaze most likely was due to the underaction of the lateral rectus that persisted. The straight eyes for near and downward gaze suggested that the under-



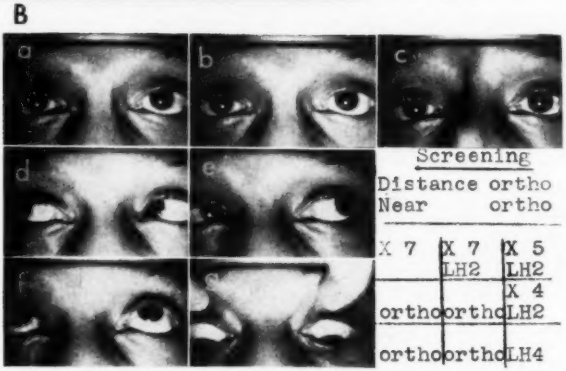


Fig. 2.—*A*, preoperative examination: (*a*) 25 degrees of right esotropia for distance, homonymous diplopia; (*b*) 20 degrees of left esotropia for near (eyes straight for near at times); (*c*) near point of convergence 50 mm.; (*d*) depression of left eye in adduction, relative elevation of right eye with no limitation in abduction; (*e*) depression of right eye in adduction, relative elevation of left eye with no limitation in abduction; (*f*) 25 degrees of right esotropia on looking up; (*g*) eyes straight looking down, stereopsis in reading position at zero.

B, postoperative examination, five months after an 8-mm. bilateral resection of the lateral rectus muscle: (*a*) eyes straight for distance, no diplopia; (*b*) eyes straight for near, stereopsis at zero; (*c*) near point of convergence remote on taking of photograph, actually 50 mm. on examination; (*d*) no depression in adduction on gaze to right; (*e*) depression in adduction of right eye on gaze to left with relative elevation of left eye; (*f*) no deviation on upward gaze; (*g*) no deviation on downward gaze.

Fig. 3.—Postoperative examination, two years after 4-mm. recession of right lateral rectus muscle: (*a*) eyes straight with glasses for distance and near with stereopsis; (*b*) 15 degrees of right esotropia without glasses for distance with homonymous diplopia; (*c*) near point of convergence 25 mm.; (*d*) slight depression of left eye in adduction, relative hypertropia of right eye, no limitation of right eye in abduction; (*e*) slight depression of right eye in adduction, relative hypertropia of left eye; (*f*) 10 degrees of right esotropia on looking up, with homonymous diplopia; (*g*) no deviation on downward gaze, with no diplopia.

action of the lateral rectus muscles had a minimal effect in these positions. Stereopsis for near may have been the important factor in preventing overaction of the medial rectus muscles, which might have produced an esotropia for near and downward gaze.

CASE 3 (Fig. 3).—C. P., aged 8 years, came to the Infirmary in January, 1949, with the history of diplopia and crossed eyes following operation on her right eye for exotropia about two years before. The diplopia was present for distance only. There was no trouble with reading. Information obtained from another hospital disclosed that before operation the child had had 15 degrees of exotropia at times. A large exophoria had been present most of the time.

With atropine cycloplegia, refraction was as follows:

O.D.: — 9.00 sph. ⊃ + 2.50 cyl., ax. 95 = 20/20
O.S.: + 1.25 sph. = 20/20

Measurements with the cover test were as follows: With glasses, 20 △ of exophoria for distance and 14 △ of exophoria for near; without glasses, 18 △ of exophoria for distance and 20 △ for near. The near point of convergence was 150 to 200 mm. Stereopsis was present on the major amblyoscope. In September, 1948, a 4-mm. recession of the right lateral rectus was done for the exophoria-exotropia.

The findings at the Infirmary in October, 1950, two years after operation, are shown in Figure 3. Without glasses there was 10 to 15 degrees of esotropia for distance and upward gaze with homonymous diplopia. For near and looking down the eyes were straight with stereopsis. Bilateral depression in adduction was seen well clinically but was difficult to photograph. Little limitation in abduction of the right eye was seen (Fig. 3*d*). The homonymous diplopia for distance was approximately the same on gaze to the right and to the left.

This patient had what appeared to be a divergence paralysis when she took her glasses off. The symptoms were due most likely to the slight paresis produced by the 4 mm. of recession of the right lateral rectus.

In Case 4 symptoms of divergence paralysis developed after bilateral recession of the lateral rectus muscle for exotropia.

CASE 4.—D. B., aged 9 years, came to the Infirmary on Jan. 6, 1949, with a history of the right eye turning out at times, especially when he was tired and when looking at something close.

With atropine cycloplegia, refraction was as follows:

O.D.: + 2.75 sph. = 20/25
O.S.: + 2.00 sph. = 20/25

The findings on Dec. 2 are shown in Figure 4 *A*. There were about 15 degrees of exotropia for distance and upward gaze and 25 degrees of exotropia for near and downward gaze most of the time. The eyes could be straight a good deal of the time, especially for distance. There was bilateral depression in adduction, which was seen well in the clinic but was difficult to show in photographs. The delayed cover test revealed an alternating hypertropia. The near point of convergence was remote. Measurements with the cover test (Fig. 4 *A*) showed that the exotropia was nonconcomitant in that the squint was greater for near and down than for distance and up, and on gaze to the left than on gaze to the right. The findings in the cover test, with the remote near point of convergence and the history of the right eye deviating out most of the time, suggested that weakness of the right medial rectus was present.

Operation, on Feb. 1, 1950, consisted of bilateral 7-mm. recession of the lateral rectus muscle. Postoperative findings, on Aug. 20, 1951, are shown in Figure 4 *B*. About 5 to 10 degrees of esotropia was present for distance and upward gaze. The eyes were straight for near with stereopsis. On looking down there was 25 degrees of exotropia. The near point of convergence was remote. The delayed cover test revealed an alternating hypertropia. Homonymous diplopia was present for distance. The separation of the images did not change on gaze to the left or to the right. The images of the light came together about 12 in. (30 cm.) from the eyes, where

there was fusion. When the light was brought closer than 9 in. (22.9 cm.) there was crossed diplopia. Postoperative symptoms of divergence paralysis developed, which might be explained as follows:

The recession of relatively normal lateral rectus muscles to correct an exotropia which was probably due to underacting medial rectus muscles resulted in a pronounced underaction of the lateral rectus muscles. This caused the esotropia and diplopia for distance and looking up. The eyes were straight for near, and there was no diplopia. This finding may be explained by the good fusion amplitude being able to balance the interplay between the weakened lateral rectus muscles,

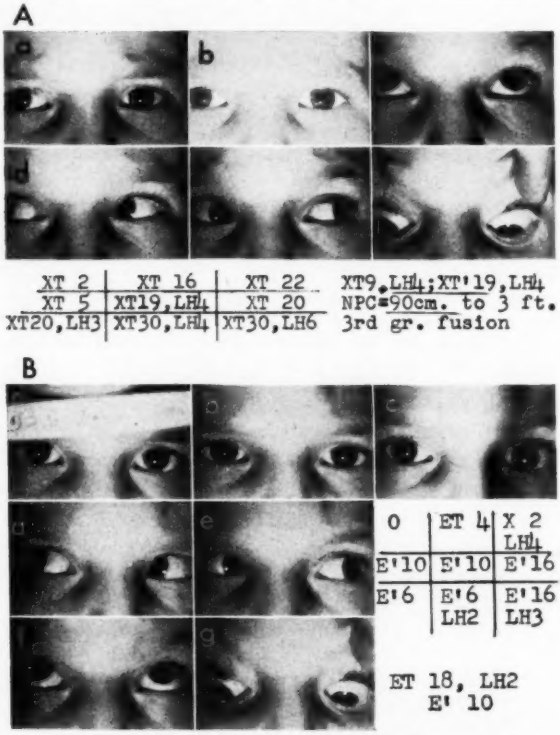


Fig. 4.—*A*, preoperative examination: (*a*) 25 degrees of right exotropia for near; (*b*) 15 degrees of right exotropia for distance, eyes can be straight for distance, stereopsis; (*c*) 10 degrees of right exotropia on looking up (eyes can be straight on looking up); (*d*) eyes level on gaze to right (depression in adduction seen clinically); (*e*) slight depression of right eye in adduction; (*f*) 25 degrees of right exotropia on looking down (eyes never straight looking down).

B, postoperative examination, 18 months after bilateral 7-mm. recession of lateral rectus muscle: (*a*) about 5 degrees of left esotropia for distance with homonymous diplopia; (*b*) eyes straight for near, with third-grade fusion and no diplopia; (*c*) remote near point of convergence; (*d*) eyes level on gaze to right, depression in adduction seen well clinically; (*e*) slight depression of right eye in adduction on gaze to left; (*f*) eyes straight on looking up, 5 to 10 degrees of esotropia possible on looking up; (*g*) 25 degrees of right exotropia on looking down.

tending to cause an esotropia, and the uncorrected underaction of the medial rectus muscles, tending to cause an exotropia, with the result that the eyes were straight in the central field.

Case 5 illustrates the presence of diplopia which simulated divergence paralysis when the eyes were dissociated by a red glass.

CASE 5.—M. J., aged 4 years, came to the Infirmary in 1934 with a history of occasional crossing of her eyes since birth. She also complained of double vision at times when looking into the distance.

Examination in 1934 revealed 15 degrees of esotropia for distance and near. With atropine cycloplegia, refraction was as follows:

O.D.: + 3.00 sph. = 20/30
O.S.: + 3.00 sph. = 20/30

The full atropine correction was prescribed to be worn all the time.

The findings on Jan. 5, 1950, are shown in Figure 5. The eyes were straight for distance and near with glasses, and the patient had stereopsis. Without glasses the eyes were straight for near with 20 degrees of esotropia for distance. The near point of convergence varied from 40 to 80 mm. The delayed cover test revealed an alternating hyperphoria. Most of the time there was no diplopia for distance. When a red glass was placed over the right eye and the patient looked

Fig. 5.—(*a*) Eyes straight for distance with glasses; (*b*) eyes straight for near without glasses, stereopsis; (*c*) 20 degrees of left esotropia for distance without glasses; (*d*) depression of left eye in adduction, relative elevation of right eye; (*e*) depression of right eye in adduction, relative elevation of left eye; (*f*) 20 degrees of left esotropia on looking up; (*g*) 5 to 10 degrees of right exotropia on looking down.

at a light in the distance, she had homonymous diplopia. The separation of the double images did not change on gaze to the left or to the right. There was slight hypertropia on gaze to the right. When the red glass was placed over the left eye, there was slight left hypertropia on gaze to the left. The double images could be fused about 15 in. (38 cm.) from the eyes. When the light was brought closer than 8 in. (20 cm.), there was crossed diplopia. Homonymous diplopia was present on looking up, while the images came together on looking down.

This patient had all the typical findings for classification in Group II. The diplopia elicited with the red glass was characteristic of a divergence paralysis. The cause of the condition may have been a congenital paresis of the lateral rectus muscles, of which presence of the esotropia for distance and upward gaze may have been a manifestation. There was no overaction of the medial rectus muscles,

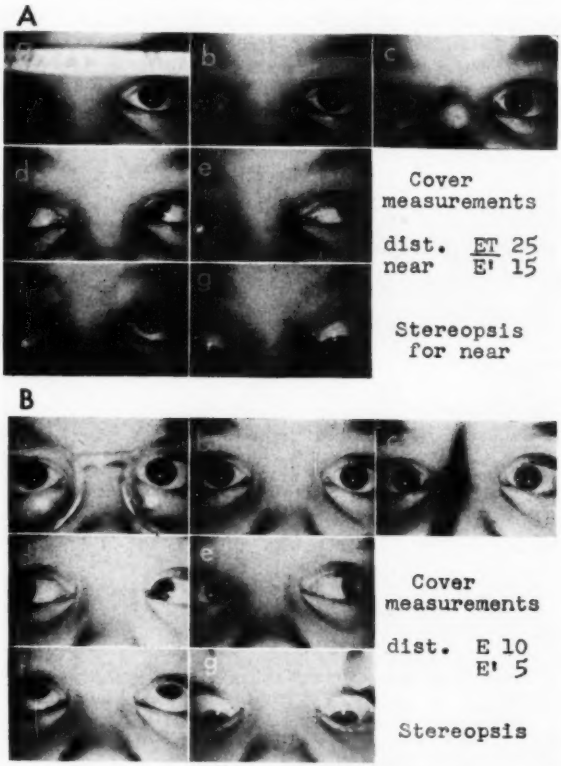


Fig. 6.—*A*, examination on Jan. 9, 1951, six months after onset of esotropia for distance: (*a*) 5 to 10 degrees of right esotropia for distance, with homonymous diplopia; (*b*) no deviation for near with stereopsis, no diplopia; (*c*) near point of convergence 35 mm.; (*d*) and (*e*), slight bilateral depression in adduction with no limitation in abduction (seen clinically, not shown well in photographs); (*f*) 10 degrees of left esotropia on looking up, with homonymous diplopia; (*g*) no deviation on looking down and no diplopia, tendency toward divergence.

B, examination Feb. 9, 1951, after glasses had been worn one month: (*a*) eyes straight for distance (and near) with glasses, no diplopia; (*b*) eyes straight for distance (and near) without glasses, no diplopia; (*c*) near point of convergence 30 mm.; (*d*) and (*e*), eyes level on lateral gaze; (*f*) eyes straight on looking up, no diplopia; (*g*) eyes straight on looking down, no diplopia.

as shown by the eyes being straight for near. In fact, a definite inhibition of the medial rectus muscles may have existed, as was suggested by the exotropia of 5 to 10 degrees on downward gaze (Fig. 5, *g*). The presence of stereopsis for near seemed to have been an important factor in preventing the overaction of the medial rectus muscles and in keeping the eyes straight.

Cases 6 and 7, in which the esotropia was considered to be of accommodative origin, are included in this paper because of the presence of diplopia simulating a divergence paralysis.

CASE 6.—R. M., aged 8 years, came to the Infirmary in December, 1950, with a history of crossing of the left eye for the past six months and of double vision for distance. His sister, two years younger, had a squint.

With atropine cycloplegia, refraction was as follows:

O.D.: + 5.00 sph. ⌒ + 1.25 cyl., ax 90 = 20/40
O.S.: + 5.00 sph. ⌣ + 1.75 cyl., ax 85 = 20/50

The findings on Jan. 11, 1951, are shown in Figure 6 *A*. There was 10 to 15 degrees of esotropia for distance and upward gaze with homonymous diplopia. The separation of the double images did not change on gaze to the right or to the left. The eyes were straight for near and looking down, when there was stereopsis and no diplopia.

The findings on Feb. 9, 1951, after the patient had worn the full atropine correction for one month, are shown in Figure 6 *B*. The eyes had become straight in all positions when the glasses were worn. The patient had no diplopia.

This case was one of an accommodative squint to a large extent, since after the correction had been worn for a short time the eyes became straight in all positions. Until the age of 8, when the squint developed, the patient was able to dissociate accommodation from convergence to a large degree, since he had a large amount of hypermetropia and yet could see clearly without glasses. The cause of the breakdown in his ability to maintain straight eyes could not be determined.

If this case actually was one of an accommodative squint as it is generally understood, it was extraordinary in that the eyes were crossed for distance and there was diplopia, whereas the eyes were straight for near and there was stereopsis. This is contrary to the usual condition in that in an accommodative esotropia there is ordinarily more esotropia for near than for distance, since more accommodation is used for near. There can be no doubt of the accommodative factor, since the esotropia was present for six months with no improvement. After the glasses were worn, the correction of the esotropia was immediate and dramatic, and the symptoms of the divergence paralysis disappeared.

CASE 7.—A. H., aged 8½ years, came to the Infirmary on March 2, 1951, with a history of the onset of acute tonsillitis two weeks before, for which she was given a sulfanilamide tablet. After this her hands became swollen, and a generalized rash developed. The mother noticed that the pupils became extremely dilated, and then the eyes became crossed. With the crossing of the eyes, the child complained of double vision being present constantly while looking into the distance.

Examination on admittance revealed 15 to 20 degrees of alternating esotropia, which was slightly greater for distance. Pseudoptosis was present on the right. Homonymous diplopia was present for distance.

With atropine cycloplegia, refraction was as follows:

O.D.: + 5.00 sph. = 20/20
O.S.: + 6.25 sph. = 20/20

The findings on April 11, 1951, immediately after the full atropine correction was worn for the first time, are shown in Figure 7 *A*. For distance, there were 15 degrees of esotropia with glasses and 20 degrees of esotropia without glasses, and the patient had homonymous diplopia. The diplopia did not vary on gaze to the left or to the right. There was 20 degrees of esotropia on looking up. For near, with glasses, the eyes were straight in the reading position, and there was no diplopia. On looking down, there was at times an exotropia, and at times, an esotropia of 5 to 10 degrees.

The findings on April 25, 1951, after the glasses had been worn for two weeks, are shown in Figure 7 *B*. With glasses the eyes were straight for distance and near, with no diplopia. Without glasses the eyes were straight for near and had 20 degrees of esotropia for distance with homonymous diplopia. The diplopia did not vary on gaze to the left or to the right. Fusion of the double images was possible about 0.5 meter from the eyes. When the fixation light was

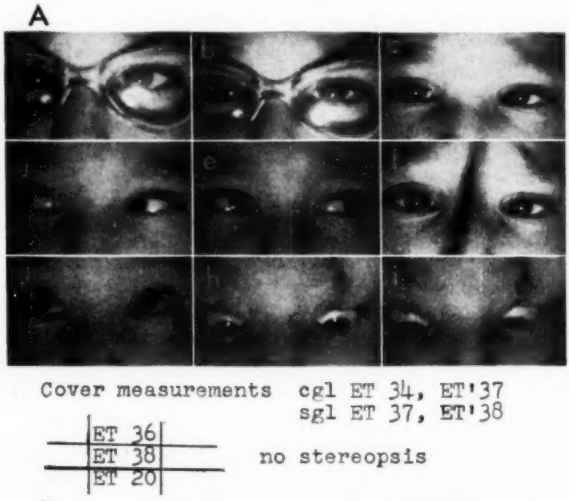


Fig. 7.—*A*, examination on day glasses were first put on: (*a*) 15 degrees of left esotropia for distance with glasses, homonymous diplopia; (*b*) 5 degrees of left esotropia for near with glasses (eyes can be straight for near with no diplopia in the reading position); (*c*) 20 degrees of left esotropia for distance without glasses, homonymous diplopia; (*d*) and (*e*), eyes level on lateral gaze (slight depression of right eye in adduction seen clinically); (*f*) near point of convergence 40 mm.; (*g*) 20 degrees of left esotropia on looking up; (*h*) slight exotropia on looking down; (*i*) 5 degrees of left esotropia on looking down.

B, examination after glasses had been worn two weeks: (*a*) eyes straight for distance with glasses, no diplopia; (*b*) eyes straight for near without glasses, stereopsis; (*c*) near point of convergence 40 mm.; (*d*) slight elevation of left eye in adduction; (*e*) eyes level on lateral gaze to left; (*f*) 20 degrees of left esotropia for distance without glasses, homonymous diplopia; (*g*) no deviation on upward gaze, 10 degrees of esotropia possible; (*h*) no deviation on downward gaze; (*i*) slightly divergent looking down.

brought closer than 100 mm., there was crossed diplopia. After the glasses had been worn for six weeks, the eyes were straight in all positions both with and without glasses. There was no diplopia, and the correction was reduced by a +1 sphere.

This child had good vision and straight eyes until she was 8½ years of age, even though she had a large amount of hypermetropia. This indicated the presence of good fusion amplitude and excellent ability to dissociate accommodation from convergence, since her eyes were always straight. After the illness and drug rash an esotropia developed. She also had symptoms of involvement of the sympathetic nervous system, i. e., mydriasis, so pronounced that the mother noticed it, and transient pseudoptosis, which was observed on the child's admission to the clinic. Involvement of the lateral rectus muscles may have been present, as indicated by the greater esotropia for distance and upward gaze. For near and downward gaze the eyes were straight a good deal of the time.

The patient's chief complaint was the constant diplopia for distance, which simulated divergence paralysis. Most of the time there was no diplopia on reading. The esotropia had a predominantly accommodative component, since glasses promptly corrected the deviation. The exotropia found on looking down (Fig. 7 *B,i*) when the eyes were straight for near may have been a sign of inhibition of convergence and of the medial rectus muscles in order to keep the eyes straight. The breakdown of the convergence-accommodation relation was on a toxic basis with possible involvement of the lateral rectus muscles and the sympathetic nervous system.

Cases 8 and 9, one a case of unilateral, the other a case of bilateral, congenital paralysis of the lateral rectus muscle, are presented to show that even with a very gross limitation in abduction the eyes could be straight for near in the central field with good fusion. Study of these cases was very helpful to me in explaining some of the findings in cases of simulated divergence paralysis. They are believed to be of sufficient interest to be included in this paper.

Case 8.—J. Z., aged 4 years, came to the Infirmary on March 28, 1948, with a history of turning-in of the left eye at times since the age of 9 months.

With atropine cycloplegia, refraction was as follows:

O.D.: + 1.25 sph. ⊂ + .50 cyl., ax 10 = 20/20
O.S.: + 3.00 sph. = 20/100

Occlusion of the right eye brought vision in the left eye up to 20/25 in three months.

The findings in June, 1950, are shown in Figure 8. The eyes were straight for distance and near in the central field. There was 10 to 20 degrees of exotropia on downward gaze, and on looking up the eyes could be straight or have about 5 degrees of exotropia at times. There were paralysis of the left lateral rectus muscle and relative elevation of the left eye on gaze to the left. The eyes were level on gaze to the right, and there was slight narrowing of the palpebral fissure, suggesting Duane's retraction syndrome. No limitation in adduction was present on gaze to the right. The near point of convergence varied from remote to 80 mm. Even with such a gross paralysis of the left lateral rectus, the eyes were straight in the central field of gaze and the patient had stereopsis. This condition indicated that there was no overaction of the left medial rectus in response to the paralysis of the lateral rectus. Actually, there was inhibition of the medial rectus (direct antagonist). This was suggested by the remote near point of convergence and the exotropia on looking down.

Case 9.—A. W., aged 4 years, came to the Infirmary in 1950 with a history of crossed eyes since birth.

With atropine cycloplegia, refraction was as follows:

O.D.: + 1.00 sph. = 20/30
O.S.: + 1.00 sph. = 20/30

The findings are shown in Figure 9. There were 20 degrees of esotropia for distance and upward gaze, no deviation for near, and 25 degrees of exotropia for downward gaze. There were bilateral depression in adduction and bilateral limitation in abduction. The near point of convergence was 100 to 150 mm. The delayed cover test showed an alternating hypertropia. The fusion status and measurements with the cover test could not be obtained because of lack of cooperation.

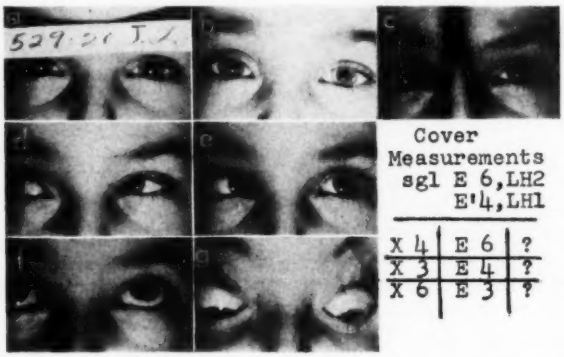


Fig. 8.—(*a*) Eyes straight for distance; (*b*) eyes straight for near, stereopsis; (*c*) remote near point of convergence; (*d*) eyes level on gaze to right, no limitation in adduction, slight narrowing of left palpebral fissure; (*e*) marked paralysis of left lateral rectus with relative elevation of left eye, widening of left palpebral fissure; (*f*) slight exotropia on looking up (eyes can be straight looking up); (*g*) 15 to 20 degrees of exotropia on looking down (eyes never straight looking down).

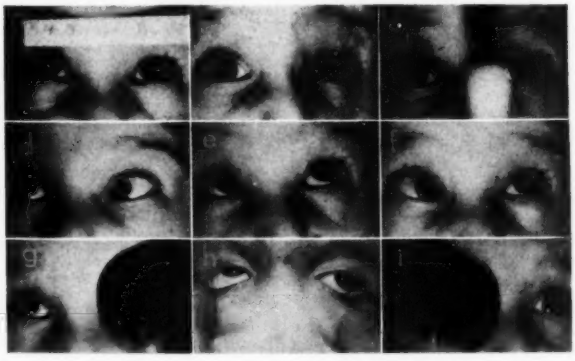


Fig. 9.—(*a*) Esotropia of 20 degrees for distance (child is looking over camera); (*b*) eyes straight for near; (*c*) remote near point of convergence; (*d*) depression of left eye in adduction, relative elevation of right eye with limitation in abduction; (*e*) 20 degrees of right esotropia on looking up; (*f*) depression of right eye in adduction, relative elevation of left eye with limitation in abduction; (*g*) limitation of right eye in abduction; (*h*) 25 degrees of right exotropia on looking down; (*i*) limitation of left eye in abduction.

This case was characterized by exaggerated findings on versional movements which were typical for classification in Group II. The pronounced bilateral limitation in abduction indicated that the cause of the condition was bilateral congenital

paralysis of the lateral rectus. The esotropia was pronounced for distance and upward gaze, a finding consistent with the presence of the underaction of the lateral rectus muscles. The straight eyes for near was unusual, since such a marked paralysis of the lateral rectus muscles was present. This finding may be explained for this case as follows:

The mechanical factor of limitation of rotation in was slight, since no gross limitation in adduction was seen. The major factor in keeping the eyes straight for near was most likely the underaction of the medial rectus muscles by means of inhibition of convergence in the interest of single binocular vision. This was demonstrated by the remote near point of convergence and the 35 degrees of exotropia on looking down, indicating pronounced underaction of the medial rectus muscles in neutralizing or balancing the 20 degrees of esotropia present on upward gaze in order to keep the eyes straight for near in the central field.

COMMENT

The cause of the simulated divergence paralysis in the cases presented was unilateral or bilateral paralysis of the lateral rectus muscle, and in two cases an accommodative component was prominent.

We have observed that paralysis of the lateral rectus muscles in general can have a pronounced effect on the positions for distance and upward gaze (more esotropia) and a minimal effect on the positions for near and downward gaze (less esotropia or straight eyes). We have also observed that the position the eyes assumed for near in the central field depended to a large extent on what the medial rectus muscles (direct antagonists) did in response to the paralysis of the lateral rectus muscles. When the medial rectus muscles became overactive, as occurred in most cases, an esotropia developed for near and downward gaze, along with the esotropia for distance and upward gaze. On the other hand, after a unilateral or bilateral paralysis of the lateral rectus muscle, no overaction could occur, or actually there was a marked inhibition of convergence and of the medial rectus muscles, which kept the eyes straight for near. It was in such cases that simulated divergence paralysis occurred. All the patients had good fusion, which must have been an important factor in inhibiting convergence in order to maintain single binocular vision. The remote near point of convergence and the exotropia on looking down were the findings which led us to believe there was underaction of the medial rectus muscles, possibly the result of inhibition of convergence, since no gross limitation or mechanical structural anomaly could be demonstrated to account for the underaction of the medial rectus muscles.

The concept that inhibition of convergence can take place in order to keep the eyes straight for near in cases in which an esotropia for distance exists seems to be supported by clinical and experimental evidence. Adler [4] stated that in many cases of divergence paralysis there is an associated convergence insufficiency.

4. Adler, F. H.: Voluntary Mechanism for Eye Movements, in Strabismus Ophthalmic Symposium No. 1, edited by J. H. Allen, St. Louis, C. V. Mosby Company, 1950, p. 76.

Experiments on the effect of alcohol (Powell,[5] Colson [6]) and anoxemia (Velhagen,[7] Adler [8]) on the dissociated position of the eyes were made on normal subjects, with the following results:

After the ingestion of alcohol by the subject, or after exposure to anoxemia in a low pressure chamber, there developed an increasing esophoria for distance and an increasing exophoria for near regardless of what the heterophoria findings were in the subject before the experiment. In some cases there developed a frank esotropia for distance with concomitant homonymous diplopia. In these cases the eyes were straight for near, with an actual exophoria. The increasing esophoria for distance was explained by Velhagen and Powell as possibly due to involvement of the lateral rectus muscles. The increasing exophoria for near might be explained by the inhibition of convergence.

Henderson and Crosby [9] found that divergence of the eyes occurred after stimulation of certain areas in the frontal lobe of Macacus mulattus. Henderson [10] believes that the divergence produced was most likely due to inhibition of convergence by these areas.

Two cases were presented that could be classified with Group II, having symptoms of divergence paralysis and a presumptive accommodative origin (Cases 6 and 7). These cases were unusual in that the accommodative squint was present for distance only. The eyes could be straight in the reading position. Since glasses promptly and completely corrected the esotropia for distance, it was assumed that this squint was largely accommodative. This assumption was contrary to the general understanding of an accommodative squint, which is usually much greater for near than for distance. These cases could not be explained satisfactorily and remained puzzling. After a reading of Duke-Elder's [11] chapter on accommodation, it was believed that the concepts of Cogan and others offered the clue to the explanation of these cases. Cogan [12] believed and presented evidence that accommodation should be divided into accommodation for distance and accommodation for near. Accommodation for near is controlled by the circular fibers of the ciliary muscle and is innervated by the parasympathetic nervous system. Accommodation for distance is predominantly controlled by the radial fibers of the ciliary muscle and is innervated by the sympathetic nervous system. The paralysis in Cases 6 and 7 may be explained by involvement of the accommodation for distance, with resulting

5. Powell, W. H., Jr.: Ocular Manifestations of Alcohol and Consideration of Individual Variations in 7 Cases Studied, J. Aviation Med. **9**:97 (June) 1938.

6. Colson, Z. W.: Effect of Alcohol on Vision: Experimental Investigation, J. A. M. A. **115**:1525 (Nov. 2) 1940.

7. Velhagen, K., Jr.: Heterophorie unter den Bedingungen des Höhenfluges, Luftfahrtmedizin **1**:344, 1937.

8. Adler, F. H.: Effect of Anoxia on Heterophoria and Its Analogy with Convergent Concomitant Squint, Arch. Ophth. **34**:227 (Sept.) 1945.

9. Henderson, J. W., and Crosby, E. C.: An Experimental Study of Optokinetic Responses, A. M. A. Arch. Ophth. **47**:43 (Jan.) 1952.

10. Henderson, J. W.: Personal communication to the author.

11. Duke-Elder, W. S.: Text Book of Ophthalmology, Vol. 4, St. Louis, C. V. Mosby Company, 1949, p. 4416.

12. Cogan, D. G.: Accommodation and the Autonomic Nervous System, Arch. Ophth. **18**:739 (Nov.) 1937.

esotropia, without the accommodation for near being affected very much. In case 7 other involvement of the sympathetic nervous system seemed to be present, as manifested by the extreme dilation of the pupils before the onset of the esotropia. The findings in these two cases are clinical evidence for the concept that accommodation for distance may be separate from accommodation for near and that a derangement of one, with its relation to convergence, can be present without the other being appreciably affected.

SUMMARY

After paralysis of the lateral rectus muscles the heterotropia in the cases presented followed an atypical course. An esotropia was present for distance and upward gaze, with diplopia, whereas the eyes were straight for near. Little limitation in abduction could be found. The paralysis in these cases resembled a divergence paralysis.

Inhibition of convergence, possibly resulting from the good fusion amplitude present in these cases, seems to have prevented the overaction of the medial rectus muscles (direct antagonists) from taking place. This could explain the eyes being straight for near.

Two cases of accommodative esotropia for distance with eyes straight for near and with symptoms of divergence paralysis are presented as clinical evidence in support of the concept that accommodation for distance may be a separate function from accommodation for near.

LAMELLAR RESECTION OF SCLERA IN TREATMENT OF RETINAL DETACHMENT

Preliminary Report

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THE PURPOSE of this preliminary report is to draw the attention of American ophthalmic surgeons to the procedure of lamellar resection of the sclera, which consists of the removal of tangential strips of three-fourths of the scleral thickness in conjunction with diathermy coagulation. The sclera is shortened by means of buried nonabsorbable (silk) surgical sutures U. S. P., causing an infolding of the surgically thinned area. The operation is simple to perform and does not expose the globe to any of the dire complications attending penetrating scleral resections. Ordinarily, the number of cases seen in any individual clinical practice is not great enough to permit the collection of a mass of statistics in a short period. Therefore it is hoped that the experience of others will be an aid to a more accurate knowledge of the indications for its use and even to improvements in technique, to the end that this procedure will fall into its proper place in the treatment of retinal separation.

Nowadays, in the so-called good cases, we may expect from 70 to 90% cures with the classic diathermy procedure. Using lamellar resection, Paufique had success in 60% of so-called bad cases, i. e., cases in which the classic operation would undoubtedly have failed. He performed 66 operations on 57 eyes.

The Müller operation of penetrating scleral resection for shortening the eyeball was revived by Lindner [1] (1933) for the treatment of retinal separation. In this country reports concerning this procedure were made, among others, by Borley, Brown, Pischel, Bogart, and Vail. However, not only because of technical difficulties but also because of many disastrous complications, the operation never gained wide acceptance in the United States. Hemorrhages, loss of solid vitreous, and, finally, rapid shrinking of the globe have not infrequently occurred. This, naturally, led many to search for a method of scleral shortening that could be done without these hazards.

Lindner [2] endeavored to shorten the eyeball by infolding a "surgically thinned" sclera but abandoned the method because the sclera unfolded and the sutures absorbed. Evidently, he employed absorbable (gut) instead of nonabsorbable (silk) surgical sutures. According to Fanta,[3] histologically, with absorbable surgical

1. Lindner, K.: Case of Retinal Detachment Cured by Eyeball-Shortening Operation, Klin. Monatsbl. Augenh. **90**:757 (June) 1933.

2. Lindner, K.: Shortening of Eyeball by Scleral Furling, Wien. klin. Wchnschr. **61**:206 (April) 1949, in Ophth. Lit., No. 665. Vol. 3, p. 182.

3. Fanta, H.: The Sclera After Lindner's Shortening Operation, Transactions of the 16th International Congress, 1950, Vol. 2, p. 1199.

sutures there is a loose cicatrization and occasionally complete dehiscence of the scleral margins, while with silk, scleral wounds heal very well indeed; silk as suture material promotes the invasion of the episcleral tissue along the suture tracks, improving cicatrization. At the meeting of the 16th International Congress of Ophthalmology, London, in 1950, Weve [4] presented a paper in which he recommended "reefing" of the sclera by means of a strong and durable material called "supermit" without resecting any of its layers. A meandering or a crossed stitch of this material is inserted through the superficial layers of the sclera over one-third or more of its circumference. The sutures are placed between the ora serrata and the equator, the anterior bites being placed from 2.5 to 3 mm. behind the ora serrata. The width of sclera to be "reefed" or folded is usually not more than 6 or 7 mm. The tension of the eyeball is lowered either by diathermic puncture or by paracentesis of the anterior chamber. He stated that the "reefing" may also be used in combination with diathermy coagulation, but he did not stress this point. In a series of 28 cases deemed unsuitable for the classic diathermy operation, the operation produced improvement in 10 and was unsuccessful in 8. According to Weve, in a number of these unsuccessful cases some improvement could have been obtained by further or by more extensive operations. I, personally, have had no experience with this technique except in one case, in which, owing to two previous operations, the sclera was so thin in one quadrant that resection was deemed inadvisable in this area. However, resection (lamellar) of the sclera was done in the contiguous area. The eye was aphakic, and no improvement resulted. This was the second case in my first series of five desperate cases.

In Lyons, in the summer of 1951, Professor Paufique discussed with me the feasibility of lamellar resection of the sclera in association with diathermy coagulation. He said that Shapland, of London, had also proposed this type of operation. Originally Paufique performed the resection without diathermy coagulation, but the results were not favorable. This spring he published his magnificent paper,[5] in which he described his personal technique, indications for the operation and results. On my return from abroad, in the autumn, I began to employ this operation. My first experiences were in five hopeless cases in which the detachments were total and had existed for over two years. Nevertheless, I was encouraged when in one of these desperate cases (the fourth), after the second part of the circumference of the eye had been subjected to a scleral resection, the patient obtained slight improvement in vision. Where formerly he had had faulty perception of light, he now had the ability to count fingers at 2 ft. (60 cm.). In this case the cure was a partial anatomic one, and no great improvement in vision or function could be expected, considering the age of the detachment. I then proceeded to employ the operation as a primary procedure in so-called bad cases, and so far cures have been obtained in five cases. Two of these were of aphakic eyes where no tears were found. In the third case secondary glaucoma followed low-grade uveitis. The pressure in this eye had been normalized by two previous operations. Two years later a detachment of the nasal half of the retina occurred. The

4. Weve, H. J. M.: Shortening of the Sclerotic ("Reefing") in Analogy with the Furling of Sails: Description of a New Technique, Indications, and Results, Transactions of the 16th International Congress, 1950, Vol. 2, p. 1179.

5. Paufique, L.; Hugonnier, R., and Moreau, P. G.: Place of Scleral Resection in Treatment of Detachment of Retina: Indications, Personal Technique and Results, Ann. ocul. **185**:113-135, 1952.

pupil was rigid, and it was impossible to find any tears ophthalmoscopically. The fourth case was that of a highly myopic eye with a large disinsertion below. The fifth was that of a 9-year-old boy in which detachment followed a perforating injury of the cornea. Except for the upper temporal quadrant, the retina was entirely detached. Below, the retina was thrown into folds, and at 6 o'clock several small cystic-appearing dehiscences, suspected of being holes, were seen.

INDICATIONS

Lamellar resection of the sclera is indicated in cases in which shrinking and retraction of the retinal-vitreous substance occur, especially when the retina has become fragile. In such cases the classic diathermy operation may result only in new tears and in more complete retinal separation. As stated by Lindner, the principle of scleral shortening lies in the attempt to adapt the sclera and the choroid to a displaced, shrunken, and shortened retina. According to Weve, shrinking and shortening of the retina occur in cases of detachment of long standing or in cases of recent detachment which is due to organization or shrinking of the vitreous or to organization on the anterior retinal surface. He stated [4]:

> In a number of cases in which the detachment was originally caused by one or more tears which seem to have been closed by previous diathermo-coagulation, a certain degree of detachment persists or develops without visible formation of new tears. In other cases tears are reopened by local shrinking of the retina secondary to the formation of so-called star-shaped folds. In some cases the persistence of a detachment is due to organization of a large hemorrhage in the corpus vitreous. Here, too, only shortening of the sclera may preserve some function to the eye.

In my opinion, in these "bad" cases, lamellar resection of the sclera should be done as a primary procedure. Eyes with high-grade myopia with giant tears or disinsertion are considered poor subjects for the classic diathermy coagulation operation. However, in many cases of myopic detachment, if one finds a typical tear and if the condition improves temporarily with bed rest, the classic procedure should be carried out. In cases of aphakia, with the well-known poor prognosis after the usual operation, I prefer the lamellar scleral resection as a primary procedure even when one or more tears can be found. In many of these cases the pupil may be rigid, or, after extracapsular operation, lenticular remains may interfere with good visualization of the periphery of the fundus. Paufique spoke of the occurrence of detachments as a result of arteriosclerosis of the retinal vessels in cases in which the retina is very fragile and numerous elongated dehiscences are found in widespread areas. Ordinarily, such detachments, and similar-appearing detachments from other causes, require extensive treatment, and even several surgical interventions, with the frequent result that the retina becomes attached in one area and contracts and produces tears in others, especially along the margin of the treated area. With lamellar resections of the sclera, the diathermy treatment used is of a much milder type than that employed in the usual operation; consequently, more extensive areas can be treated without danger. Paufique also recommended lamellar scleral resection in cases in which detachment of the retina occurs with cataractous changes in the lens which preclude accurate viewing of the fundus.

In instances in which relapses occur after the classic procedure, lamellar scleral resection can be done, but with greater difficulty. Although possible, it is admittedly more arduous to perform a lamellar resection of the sclera in an area already

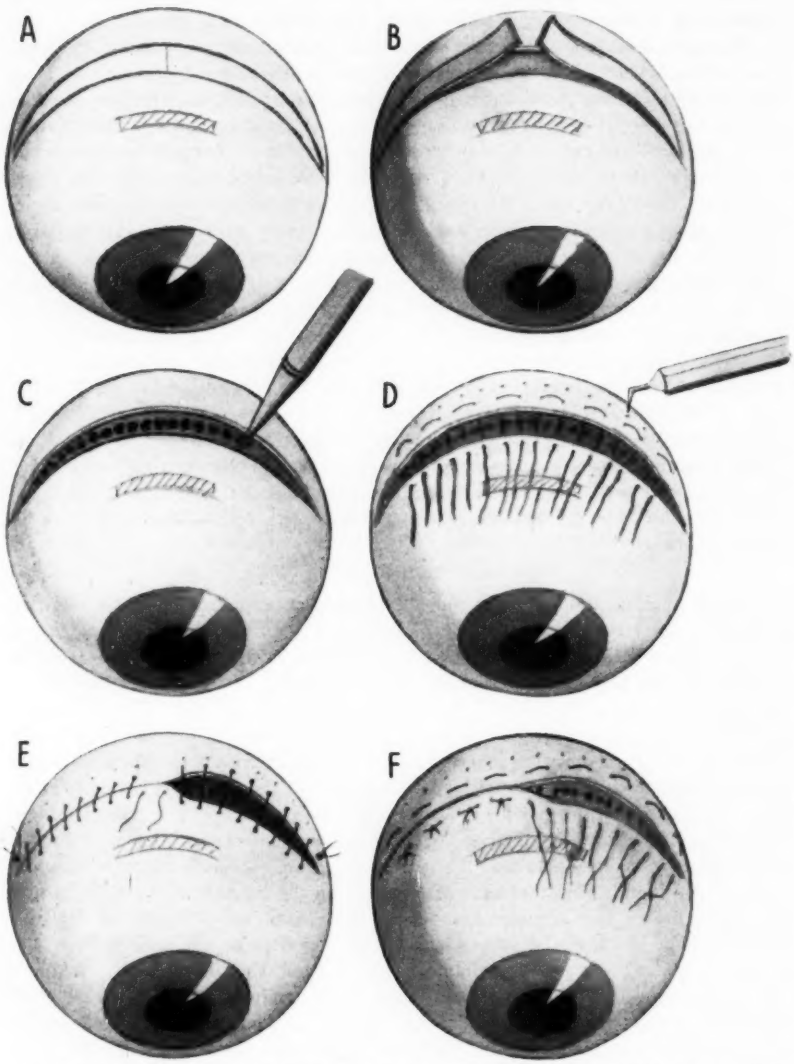
thinned by previous coagulations. Perhaps reefing (Weve) combined with diathermy may offer an easier solution in the approach to such areas.

As emphasized by Paufique, the aim of this operation, as in the classic diathermy coagulation, is the closure of the tears, a dictum insisted upon by Gonin. It seems that the milder, but more extensive, diathermy coagulations, together with the shortening effect of lamellar resection of the sclera, can accomplish this in many of the so-called bad cases. It is in these cases that the unfavorable reaction occurs to the more violent and destructive treatment necessarily required by the classic operation. As to the site of the operation for lamellar scleral resection, one should be governed by the same indications as in the classic procedure. It should be performed in the regions and at the level of the tears or, in the absence of visible tears, where the detachment seems most prominent. It is my impression that in the so-called bad cases tears are predominantly found in the far periphery. In the cases in which I operated successfully when no tears could be found, the lamellar resection of the sclera was performed from 3 to 4 mm. behind the insertion of the rectus muscles. An intelligent patient may help the surgeon if he recalls which part of the visual field was first affected by photopsias and blurring of vision. It is always wise, when one is employing lamellar resection of the sclera as a primary procedure, to warn the patient that the operation may have to be performed in stages, since some may require complete circumferential resection. The latter, preferably, should be divided into two or more sessions, with at least a two-months' interval between them.

TECHNIQUE

In my opinion, the length of the scleral strip excised should not be less than a third of the circumference of the globe; in most of my cases I have tried to remove almost half the circumference.[6] This frequently may be done with the severing of one of the tendons of the rectus muscles. Of course, depending on the tears, even two of the muscle tendons may have to be severed. The conjunctiva and Tenon's capsule over the operative areas are dissected freely. With the scleral field exposed, the strip to be removed may be outlined with a toothpick dipped in methylrosaniline chloride solution. With experience, this step may be superfluous. The strip is then outlined with a Lundsgaard knife, and about one-third to one-half the scleral thickness is incised. The anterior edge of the strip is ordinarily placed about 2 to 4 mm. behind the muscle insertion, especially in cases in which no tears are found. In other cases, depending on the location of the tears, it may be placed nearer the equator. However, the vortex veins should be carefully avoided. The width of the strip which I have found to be adequate is from 3 to 2 mm. (Fig., *A*). It has been found easier to begin the dissection by making a cut across the center of the outlined strip. One carefully deepens this cut until about two-thirds of the scleral thickness is reached. When doing this, one is guided by the darker appearance of the depth of the incised sclera as the lamina fusca is approached. One then grasps one of the edges (Hess iris forceps or the scleral forceps of Saint-Martin) and begins to dissect up the strip. Once started at the proper level, the strip comes off readily (Fig., *B*). However, one should be careful to keep the dissection at the same level to avoid perforation. In one of my early attempts this complication occurred but did not greatly interfere with the

6. The resection of a 3-mm. circumferential strip reduces the volume of the ocular contents by 12%.



A, stump of resected superior rectus tendon and marking out of scleral strip to be removed. *B*, beginning of dissection of lamellar scleral strip. The deepest remaining scleral layer appears slate-colored. *C*, coagulation treatment by gentle application of ball electrode to surgically thinned sclera. *D*, braided white nonabsorbable (silk) surgical sutures U. S. P. in place. Perforation was made with 1.5 mm. diathermy needle. *E*, alternate type of continuous suture. *F*, scleral edges of resected strip drawn together and sutures tied after escape of subretinal fluid.

progress of the dissection. The bottom of the trough appears slate-colored. After one-half is dissected, the other half is likewise resected. The edges of the scleral defect may be undermined for 1 mm. or so. The dissection having been completed, the bottom of the trough is treated with the ball electrode in a continuous way, as shown in Figure, *C*. The current should be light, about 30 ma., and applied so that the thin remaining floor is not completely destroyed. Sutures (braided 000 white silk) are then inserted either in mattress form (Fig., *D*) or in a continuous manner, as shown in Figure, *E*. With a 1 or 1.5 mm. perforating diathermy needle single punctures are made slowly about 1 to 2 mm. behind the excised strip, so as to permit escape of the subretinal fluid. Further escape of fluid can be expedited by massage of the globe, or perhaps by enlarging one of the punctures with a probe, care being taken not to push the dilating probe deeper than 1.5 mm. Upon escape of the subretinal fluid, the globe softens and makes easy the approximation of the dissected scleral edges and the infolding of the thinned-out scleral floor (Fig., *F*). The detached muscle or muscles are reattached, and the conjunctiva is closed with absorbable surgical sutures. Atropine is instilled, and both eyes are bandaged.

AFTER-TREATMENT

In the main, the after-treatment differs in no way from that employed with the classic operation. Inspection of the eyes ordinarily is not begun until after the fifth day, and then it is done every third day. The eye operated on is kept atropinized. The patient becomes ambulatory after the third week. In correspondence with Paufique's observations, I have noted that in some cases reattachment takes a little longer after lamellar resections of the sclera than in favorable cases in which the classic procedure has been done. In one of my cases the outcome was doubtful until after the third week. Only at this time did it become apparent that complete reattachment of the retina had occurred. Paufique thinks that this delay in some cases is due not to the resection but, rather, to the fact that this operation is performed in unfavorable cases and that a prolonged time for reapplication would likewise occur in such cases with any type of operation.

CONCLUSIONS

Lamellar resection of the sclera is recommended in the cases of retinal detachment in which hitherto even before operation the prognosis has been considered poor with the usual classic diathermy operation. In these cases the resection should be done as a primary procedure. Although successes have been reported when it was employed as a secondary or tertiary operation following failures with the classic procedure, it is understandable that in many such cases one may have to be satisfied with an anatomic improvement, since the retina may be so damaged as to preclude any remarkable functional result.

In cases in which doubt may arise as to which operation should be the primary one it is suggested that when binocular bandaging and bed rest for a few days do not produce clinical improvement, lamellar scleral resection with diathermy coagulation should unhesitatingly be the operation of choice.

Finally, although the time element in this operation is somewhat longer and the technique more difficult than the classic procedure, it is apparently harmless since as yet no extraordinary complications have arisen.

57 West 57th St.

BILATERAL OCCIPITAL-LOBE INFARCTION SIMULATING RETROBULBAR OPTIC NEURITIS

Report of Case Terminating Fatally During Use of Corticotropin

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THIS CASE is reported [1] because of two features which presented: first, a complication from the use of corticotropin and cortisone, namely, increased coagulability of the blood, and, second, bilateral absolute pericentral scotoma, without peripheral field defect, due to suprachiasmal, rather than prechiasmal, disease of the optic pathway.

REPORT OF CASE

A man aged 47 was admitted to the Veterans Administration Hospital, Dec. 22, 1950, complaining of progressive loss of vision in each eye during the past 10 days. The only accompanying symptom was bilateral occipital headache. Vague numbness of the right side of the face had been present for one day but had not recurred. The past history contained nothing of importance. The patient had had no acute or chronic inflammatory or vascular disease. During the past five years, in his employment as an automobile mechanic, he stated that he had been occasionally exposed to lead fumes.

On admission, his temperature, pulse, and respiration were normal. The blood pressure was 140/90. He weighed 188 lb. (85.3 kg.). The physical findings, including the electrocardiogram, were normal. Roentgenograms of the skull, with special views of the optic foramina and sella turcica, showed nothing abnormal. No neurologic change, except that evidenced by the visual complaint, was found.

Ophthalmologic examination showed that vision in each eye was reduced to light perception peripherally. A central scotoma of more than 20 degrees in extent was present in each visual field. The pupils were slightly dilated, equal, regular, and normal in reactions. Function of the extraocular muscles was normal. Nystagmus was not present. The optic fundi were normal.

The results of routine urinalysis were normal. Nonprotein nitrogen, total serum protein, albumin-globulin ratio, blood chlorides, fasting blood sugar, and carbon-dioxide combining power were normal. The serologic agglutination tests gave normal results. The spinal fluid was under a pressure of 190 mm. and contained 10 white cells and 3,000 red blood cells per cubic millimeter. The clinical diagnosis of acute retrobulbar optic neuritis was made, and treatment with parenteral administration of corticotropin was begun. Cortisone was not available at this time. Beginning on Dec. 23, 10 mg. of corticotropin was administered every six hours. The dose was increased to 15 mg. on Dec. 26 and to 20 mg. on Dec. 28. On Dec. 31 it was decreased to 15 mg., and on Jan. 2, to 10 mg. On Jan. 3 it was reduced to 10 mg. every 12 hours, and finally, on Jan. 4, to 5 mg. twice a day. At the end of the first week of treatment, Jan. 1, the patient was able to count fingers and to see color. No systemic changes had been observed. Apparently, he was responding well to treatment. That morning, paralysis

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. The case report was taken from the clinical records of the Veterans Administration Hospital.

and numbness of the left arm and leg, weakness of the left side of the face, ptosis of the left upper eyelid, and paralysis of the left internal rectus muscle appeared simultaneously. He complained of bitemporal headache. He was found to have a cloudy sensorium, with mild aphasia. Hoffmann and Babinski reflexes were elicited on the left side. Apparently, he had suffered a cerebrovascular accident, probably thrombosis in the region of the pons. There was no change in his general condition until the fourth day after the appearance of the paralyses, when the paralysis of the left facial nerve was increased. On the fifth day he became comatose, and on the sixth he died.

Autopsy.—A complete examination was performed. The eyes, optic nerves, and optic chiasm appeared normal grossly and microscopically. The other organs of the body were essentially normal except for the brain. An excerpt from the report follows.

"The arteries at the base showed fairly severe, 3+ arteriosclerosis. The optic chiasm and the optic nerves and tracts appeared normal. Coronal sections after preliminary fixation revealed nearly symmetrical lesions in the occipital lobes; these lesions involved the posterior poles and the medial aspects, including the visual area of both lobes, and were characterized by softening and discoloration of the cortex and subcortical white matter with disseminated punctate hemorrhages throughout the necrotic tissue. Elsewhere there were no obvious gross lesions.

Microscopic examination of routine sections of brain showed nothing of note except for intense congestion and edema. The sections from the calcarine and occipital areas showed varying states of necrosis, due to repeated vascular thrombosis. Sections from these areas showed, in places, small vessels to be widely dilated and to contain old and fresh thrombi. The brain substance in areas adjacent to vessels of this type showed extreme encephalomalacia and destruction with a gitter-cell reaction. In other areas, where the vessels had been occluded by fresh thrombi only, the vessel walls were undergoing necrosis, and the adjacent brain substance showed early necrosis with a marked leucocytic reaction. The section from the occipital area also showed perivascular cupping with small round cells. All cerebral vessels seen in these sections showed severe arteriosclerotic changes, with formation of plaques, and many contained old and fresh thrombi."

COMMENT

The abrupt appearance of a cerebrovascular accident in this patient, who was receiving treatment for acute retrobulbar optic neuritis, caused a rapid reconsideration of the original diagnosis. The possibility of an unusual reaction to corticotropin was discussed. It was suggested that the patient's natural tendency to vascular thrombosis associated with his arteriosclerosis might have been exaggerated by his treatment. The first published report on the hypercoagulability of the blood associated with corticotropin and cortisone therapy appeared coincident with the period of this patient's illness. Other reports have since been published. Unfortunately, no coagulation studies were made upon this patient's blood, so that one can only reflect whether or not the corticotropin he received had any deleterious effect. Autopsy demonstrated that he had had an occipital cerebral thrombosis before his treatment and that his death was probably from thrombosis of vessels of the brain stem, which occurred one week after he had begun to receive corticotropin therapy. Whether or not he would have had this second vascular thrombosis if he had gone untreated we cannot know.

Cosgriff [2] has recently stated that "hypercoagulable changes observed in the clotting mechanism may develop as soon as 48 hours following start of hormonal therapy and persist for as long as three weeks following discontinuance of treatment with these agents." His report showed that thrombosis is commoner in patients

2. Cosgriff, S. W.: Thromboembolic Complications Associated with ACTH and Cortisone Therapy, J. A. M. A. **147**:924, 1951.

with underlying vascular disease or with a history of previous thrombotic disease, such as our patient had had, unknown to us. Cosgriff believes that if hormonal therapy is necessary for such a patient it should be accompanied by the administration of anticoagulants.

The clinical diagnosis of retrobulbar optic neuritis was made because of the history of rapid reduction of central visual acuity, the presence of central scotomas, and the absence of any lesions visualized with the ophthalmoscope. The age and sex of the patient and the essentially normal physical, x-ray, and laboratory findings, except for slight hypertension, further contributed to an arrival at this diagnosis. Autopsy revealed that this was an incorrect assumption but that, in fact, the signs of retrobulbar optic neuritis were explainable only by the presence of simultaneous infarctions developing at the posterior poles of the occipital lobes.

OCULOGRAVIC ILLUSION

CAPTAIN ASHTON GRAYBIEL (MC), U.S.N.

DURING the past four years numerous experiments have been carried out which had in common the fact that a person subjected to a change in direction of resultant force relative to himself experienced an illusion. This consisted partly of a feeling of being tilted and of assuming a new position in space and partly of an impression that objects in the visual field moved and occupied new positions in space. These changes accorded more or less with the change in direction of the resultant force. The visual component of this phenomenon was called the "oculogravic illusion," and time has proved the usefulness of this descriptive term.

Although this illusion has been known since the time of Mach,[1] it has not been the object of intensive investigation. No attempt will be made to summarize past work in this field, since this has already been reviewed adequately in other places.[2] Previous reports from this laboratory [3] have dealt with systematic studies of certain features of this illusion, whereas the present report is of a more qualitative character and includes many observations hitherto unpublished.

EXPERIMENTAL PROCEDURE

All the experiments to be reported upon were carried out on healthy adult subjects of both sexes who were thoroughly familiar with the procedure. They were exposed to centripetal

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1. Mach, E.: Grundlinien der Lehre von den Bewegungsempfindungen, Leipzig, Wilhelm Engelmann, 1875.

2. Boring, E. G.: History of Experimental Psychology, New York, D. Appleton-Century Company, Inc., 1942. Dusser de Barenne, J. G.: The Labyrinthine and Postural Mechanisms, in A Handbook of General Experimental Psychology, edited by C. Murchison, Worcester, Mass., Clark University Press, 1934, pp. 204-246. Griffith, C. R.: Historical Survey of Vestibular Equilibration, Univ. Illinois Bull. **20**:1-178, 1922. McNally, W. J., and Stuart, E. A.: Physiology of Labyrinth Reviewed in Relation to Seasickness and Other Forms of Motion Sickness, War Med. **2**:683-771, 1942. Witkin, H. A.: Perception of Upright When Direction of Force Acting on Body Is Changed, J. Exper. Psychol. **40**:93-106, 1950.

3. (*a*) Graybiel, A.; Clark, B., and MacCorquodale, K.: Illusory Perception of Movement Caused by Angular Acceleration and by Centrifugal Force During Flight: I. Methodology and Preliminary Results, J. Exper. Psychol. **37**:170-177, 1947. (*b*) Clark, B.; Graybiel, A., and MacCorquodale, K.: IV. Illusory Rotation of a Target During Turns, Am. J. Psychol. **61**:50-58, 1948. (*c*) Clark, B., and Graybiel, A.: Apparent Rotation of Fixed Target Associated with Linear Acceleration in Flight, Am. J. Ophth. **32**:549-557, 1949. (*d*) Graybiel, A., and Brown, R. H.: Delay in Visual Reorientation Following Exposure to Change in Direction of Resultant Force on Human Centrifuge, J. Gen. Psychol. **45**:143-150, 1951. (*e*) Clark, B., and Graybiel, A.: Visual Perception of Horizontal Following Exposure to Radial Acceleration on Centrifuge, J. Comp. & Physiol. Psychol. **44**:525-534, 1951.

acceleration on a human centrifuge of the inertia wheel type.[3d] It consisted essentially of a rotating platform 6 ft. (180 cm.) wide and 22 ft. (6.6 meters) from center to periphery. It was suspended above a motor-driven flywheel weighing 22 tons (10 metric tons), to which it could be engaged by means of a clutch.

The subject sat in a padded seat 17 ft. (5 meters) from the center of rotation. Heavy restraining straps prevented body movement, and a biting board was used to minimize head movement. The seat could be rotated about its vertical axis and secured in one of four different positions. In the first position the subject faced toward the axis of rotation of the centrifuge; to assume the second, third, and fourth positions, the seat was rotated successively through 90 degrees of arc.

Most of the experiments were carried out in the dark with the subject fixating a dimly illuminated collimated star or a single collimated line of light.[4] In some experiments the centrifuge chamber was brightly illuminated. This room was circular, and the subject, seated on the centrifuge platform, was approximately 6 ft. away from the wall at the nearest point.

In these experiments three very important variables were controlled, namely, the force of acceleration, the position of the subject in relation to the resultant force, and the visual framework. These details are discussed later in the appropriate places. It is convenient to mention

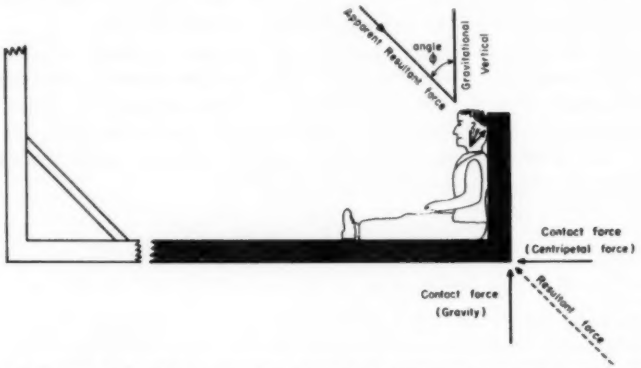

Fig. 1.—Change in direction of resultant force and "apparent" resultant force in relation to subject on human centrifuge.

here, however, the concept of "apparent resultant force."[5] This can be explained with the aid of Figure 1, which shows a person on a horizontal centrifuge exposed to a centripetal force equal to the force exerted by gravity. The resultant force is indicated by the dotted arrow (*RF*), which is the vectorial sum of the forces pressing on the subject from below and behind. This resultant force is a vector quantity which has magnitude, direction, and sense. The magnitude is expressed in units of the force of gravity. The direction in space may be indicated by the angle (ϕ) which it forms with the gravitational vertical. The sense of the vector may be shown graphically by an arrow which indicates the direction of application of the force. The resultant force (*RF*) is a "contact" force, and, although it produces its maximal effects at the points of contact between subject and restraining structure, it is transmitted to the remainder of the body through tensions and pressures developed by deformation. Sensory elements responsive to such tensions and pressures are thereby stimulated, and the subject feels that he is pressed downward and backward.

4. Clark, B., and Graybiel, A.: A Device to Manipulate and to Indicate the Position of Remote Test Objects in Studies of Visual Space Perception, U. S. Naval School of Aviation Medicine, Project NM 001 059.01.26, Research Report, Dec. 6, 1951.

5. This concept was presented before the annual meeting of the Aero Medical Association in 1950, and will form part of a forthcoming report by Mr. Frederick Dixon and Dr. John L. Patterson Jr.

Although this resultant force is basically responsible for the relative displacement of tissues within the body, these displaceable elements behave as if they were acted upon by an action-at-a-distance force which is equal but opposite in direction to the resultant force. This may be termed the "apparent" resultant force and is indicated in Figure 1 by the solid arrow, *ARF;* the action of this force in deforming the hair of a sensory cell of the otolith organ is shown diagrammatically.

The vectors *RF* and *ARF* differ only in sense. The former represent the situation with the earth considered as the basic frame of reference. When the rigid structure of the body constitutes the basis for reference, the *ARF* vector is used. It is this frame of reference which is used by the subject and hence has the more important meaning here. For convenience, the *ARF* vector is expressed as the angle (ϕ) which it makes with the gravitational vertical.

It is necessary to mention two complicating illusory phenomena which were encountered in the course of the experiment on the oculogravic illusion, namely, the autokinetic sensation and the oculogyral illusion.

The autokinetic sensation consists of a rather aimless wandering of an object, without obvious cause, when viewed against a uniform background. This illusion is not prominent when one is using a collimated star because it forms a good two-dimensional frame of reference.[6] Furthermore, subjects familiar with the autokinetic illusion and the oculogravic illusion can almost always distinguish between the two on the basis of their characteristic behavior.

The oculogyral illusion is due to the nystagmus produced when the semicircular canals are stimulated by angular acceleration.[7] In the present experiments angular accelerations were produced whenever there was a change in rate of rotation of the centrifuge platform. This angular acceleration could be kept small by changing the rate of rotation very slowly and, of course, was not present when the centrifuge rotated at constant speed. Under the circumstances of this experiment, the oculogyral illusion was seen as an apparent motion of the target in the horizontal plane in a clockwise or counterclockwise direction. The sophisticated subject could always distinguish this illusion from the oculogravic illusion.

GENERAL DESCRIPTION

A striking demonstration of the oculogravic illusion can be made in the following manner: The subject is seated facing the center of rotation, fixating a collimated star directly before him in an otherwise dark room. When all is in readiness, the subject is exposed to a change in direction of resultant force of 45 degrees in approximately three seconds. After the initial jerk the subject perceives an apparent change in body position, an apparent tilting of the supporting structures, and an apparent movement of the star.

The apparent change in position consists of a curious feeling of being tilted smoothly, yet firmly, backwards. The axis of rotation is vaguely perceived as being in the region of contact with the seat, inasmuch as the trunk seems to be inclined backward and the legs forward. This apparent shift in position takes place rather rapidly at first, then more slowly, until finally the subject is unaware of further change; it extends over a much longer period than that required to induce the acceleration. There is usually a fairly good correspondence between the subject's estimate of the degree of tilt and the angle (ϕ).

In addition to feeling that he has tilted backward, the subject also feels that the structures supporting him have tilted similarly. This applies to the seat, arms, and back of the chair, the floor under the feet, and even the biting board in the mouth.

6. Graybiel, A., and Clark, B.: Autokinetic Illusion and Its Significance in Night Flying, J. Aviat. Med. **16**:111-151, 1945.

7. Graybiel, A., and Hupp, D.: The Oculo-Gyral Illusion: A Form of Apparent Motion Which May Be Observed Following Stimulation of Semicircular Canals, J. Aviat. Med. **17**:3-27, 1946.

Coincident with these perceptions, the subject is aware that the star, previously considered to be at or near the horizon, apparently rises; it is in the nature of a smooth glide, without any jerkiness. This rise takes place rather rapidly at first, then more slowly, until finally the star comes to rest. The apparent displacement of the target in space is in keeping with its apparent rate of motion. There is little or no overshoot, the target remaining motionless at the completion of its upward movement. The time required for the ascent of the star is many times the three-second period during which the acceleration was induced. The subject's estimate of the degree of the displacement of the star above the horizon bears a good relation to the angle between resultant force and direction of gravity.

If the experiment is altered in one respect, namely, that the subject's eyes remain closed until a minute has elapsed after acceleration, he notes, on opening the eyes, that the object is now displaced and stationary; no apparent movement is observed. So long as the centripetal force is maintained at a constant level, the apparent position of the star remains the same. The definition of the target does not change; there is no blurring of the lines, and it appears to be motionless. The illusion is so compelling that neither the knowledge of the actual position of the star nor an intellectual effort of any kind will abolish it. If the centrifuge is now brought to a stop, the star appears to descend, rapidly at first, then more slowly, and sometimes a small amount of overshoot is observed.[8d]

This observation indicates that during an increase or a decrease in centripetal force an object stationary with respect to the subject appears to move in a smooth, orderly fashion. If the change in acceleration occurs suddenly, there is a considerable lag before the completion of the apparent movement, and there is little or no overshoot. At a constant speed of revolution the object appears to be motionless and to remain constantly displaced from its original position. There is a close correspondence between the magnitude of the displacement and the angle (ϕ).

AFTER-IMAGES

The following experiment differs from that described above in one particular. A strong after-image was induced by having the subject fixate a long incandescent light filament immediately prior to centrifugation. This after-image was superimposed on the collimated star, and, with the onset of acceleration, both appeared to rise. On deceleration, both images appeared to descend to the horizon.[8] The experiment was repeated using only the after-image as a target, and a similar rise and fall were observed.

The results of this experiment offer strong proof that the oculogravic illusion is not due to eye movements. If eye movements were the cause of the apparent displacement of the visual targets, the after-image would be displaced in one direction and the real image in the other, for the following reasons: If an after-image is induced and the eyes are moved, the after-image appears to move in the same direction. The reverse is true in the case of a real image; movement of the eye causing a tracking of the image over the retina in one direction is interpreted as movement of the object in the opposite direction.

8. A slight separation of after-image and real target in the horizontal plane may be observed during the apparent rise and fall of the targets. This is due to rotation nystagmus caused by angular acceleration.[7]

EXPERIMENTS IN A LIGHTED ROOM

In these experiments the room was brilliantly lighted, and, instead of fixating the star, the subject regarded the horizontal centrifuge platform and the vertical walls of the circular room. On being exposed to gradually increasing centripetal acceleration, the subject perceived the platform as horizontal and the walls as vertical until the change in direction of resultant force was 10 to 15 degrees.

When the change in direction of resultant force reached a level of about 56 degrees, an extraordinary change occurred. The platform suddenly appeared to tilt upward and become stationary like a coin on edge. The subject then regarded himself as motionless and lying on his back. The walls of the room then appeared like an endless band rotating around the centrifuge platform. This phenomenon is not unlike that which the aviator experiences when he goes into a spin.

The result of this experiment threw some light on the rivalry between visual stimuli and stimuli related to change in resultant force. The former remained constant, or nearly so, throughout, whereas the latter increased in magnitude with increasing change in direction of resultant force. At low levels the rivalry was resolved in favor of visual stimuli, inasmuch as the platform appeared horizontal and the walls vertical; at slightly higher levels the resolution favored the nonvisual stimuli, and when the centripetal force was sufficiently great, visual stimuli were overwhelmed.

CHANGE IN BODY POSITION

In these experiments the position of the subject was varied. The illusory effects perceived when the subject sat facing the center of rotation have been reported, and this description will not be repeated.

Subject Facing Away from Center of Rotation.—In this position the subject sat facing the periphery of the centrifuge platform, i. e., with his back toward the center of rotation. When suddenly subjected to a change in direction of resultant force of 45 degrees, he felt that he was tilting forward and that the seat and platform sloped downward toward the periphery. The star appeared to descend and finally to come to rest far below its original position. These effects were essentially the reciprocal of those observed when he was facing toward the center of rotation.

Subject Facing Toward Direction of Rotation.—In this position the subject sat facing ahead with respect to his movement in space; the center of rotation was now on his left. With the onset of centripetal force the subject felt that he was tilting to the right. The arm, hip, and foot felt low on the right and high on the left. The star appeared to rotate in a clockwise direction; when the apparent movement had ceased, the horizontal lines of the star appeared to be displaced approximately 45 degrees.

A variation of this experiment was carried out in which the subject sat with body facing the center of rotation but with head rotated 90 degrees toward the right, thus facing in the direction of rotation. On being subjected to centripetal force, he had a feeling of being tilted backward, but the star appeared to rotate in a clockwise direction.

Subject Facing Away from Direction of Rotation.—In this position the subject faced away from the direction of his movement in space, and the center of rotation was on his right. When subjected to a centripetal force, he felt that he was tilting

toward the left and that the seat and platform were sloping down on that side. The star appeared to rotate counterclockwise until the horizontal line had rotated through an angle of approximately 45 degrees.

Unusual Body Positions.—In these experiments the subject could be placed in any desired position in space by means of a seat mounted on gimbals. Since certain positions, especially the head-down position, were uncomfortable, the subject was exposed to centripetal forces which were sufficient to produce only a small illusory effect. Although it was possible for the subject to observe the oculogravic illusion in any body position, there were great differences with regard to the amount of centripetal force required. In general, the more unusual the body position, the greater the centripetal force necessary. It was not established whether this variation was due to differences in the behavior of sensory organs, notably the otolith organ, or to differences in the ease with which the subject could judge verticality and horizontality in unfamiliar positions.

The results of these experiments may be generalized as follows: The oculogravic illusion may be observed regardless of the position of the subject in space or of the position of the head in relation to the trunk. The apparent displacement of an object can be predicted from a knowledge of body position in relation to resultant force and the position of the head in relation to the trunk. The illusion is more readily perceived in some body positions than in others.

Oculogravic Illusion as Function of Direction of Resultant Force in Relation to Subject.—A number of experiments were performed in which the subject's estimate of the angular displacement of an object (estimated angle ϕ) was compared with the change in direction of resultant force (actual angle ϕ). Favorable conditions were provided both with regard to the perception of the illusion and the estimation of the magnitude of the illusory effect. The subject sat with the center of rotation either on his right or on his left side; thus, the change in direction of resultant force occurred in the frontal plane, and the illusion would be perceived as a clockwise or counterclockwise rotation of the object.[5e]

The fixation object usually consisted of a single line of collimated light which was observed in an otherwise dark compartment. This line could be rotated clockwise or counterclockwise about its center either by the subject on the centrifuge or by the observer in the control room. A record was obtained of the change in position of the line, and this could be compared with a record of the change in direction of the resultant force.

Threshold Measurements.—In these experiments [9] the subject was exposed to a very gradual increase in centripetal force and was signaled when he first observed clockwise or counterclockwise rotation of the line which he previously had adjusted to the horizontal. The subject was prevented from learning whether the center of rotation of the centrifuge was on his left or on his right; hence he did not know whether the line would rotate clockwise or counterclockwise.

The actual angle ϕ at which the subject reported rotation ranged from a few seconds to 4 degrees. The percentage of correct responses increased progressively

9. Brown, R. H.: Imus, H.; Niven, I. J., and Graybiel, A.: Relationship Between Apparent Displacement and Motion in the Oculogyral Illusion, U. S. Naval School of Aviation Medicine and Tulane University, Pensacola, Fla., Project No. 001 037, Joint Report 1, May 1, 1949.

with increase in the angle ϕ. When ϕ was 1 degree or less, approximately 50% of the responses were correct; when 2 degrees, 75% were correct, and when 3 degrees or more, 100% were correct.

Apparent Displacement of Object in Visual Field Compared with Change in Direction of Resultant Force.—The object of these experiments was to compare, under the most favorable circumstances, the subject's estimate of the apparent displacement of an object with the change in direction of resultant force to which he was subjected. The subject was seated with the center of rotation of the centrifuge on his left. He fixated a collimated line of light in the dark. The line could be rotated clockwise or counterclockwise by the push or pull of a lever which activated a small motor. This made it possible for the subject to "correct" for an apparent rotation of the line. Thus, during exposure to centripetal force, the arc through which the subject rotated the line, in order to maintain it at what seemed to him to be the horizontal position, could be compared with the actual angle ϕ. Various magnitudes of centripetal force were used but none was greater than that which produced a change in direction of resultant force of 45 degrees. The exposure was brought about slowly in an effort to reduce the "lag" effect, which will be described later.

Under control conditions, i. e., before exposure to centripetal force, the subject was able to adjust the line to the horizontal position with great accuracy. When exposed to a gradual increase in centripetal force, most subjects rotated the target line through an arc which closely corresponded to the actual angle ϕ. If this was succeeded by a period in which the centripetal force was constant, the correspondence between the actual and the estimated angle ϕ was similarly good. However, much individual variation was observed; in some instances the subject had difficulty in deciding when the line appeared to be horizontal and consequently rotated the target through an arc much smaller or greater than the actual angle ϕ. During periods of exposure to constant centripetal force, if the target line was offset and the subject was required to reset it, he was not as consistent in his estimate of the apparent horizontal as in that of the true horizontal under control conditions.[3e]

Adaptation.—A series of experiments were carried out to determine whether adaptation occurred during relatively prolonged exposures to centripetal force. The procedure was essentially the same as that described in the section above. The results indicated that the subject's threshold for perception of the oculogravic illusion did not change after exposure to centripetal forces of different magnitudes (maximum, $\phi = 36$ degrees) for 15 minutes, and that during these periods of constant revolution no change was observed in the subject's estimate of the apparent horizontal. Thus, it was concluded that significant changes which could be attributed to adaptation did not occur.

Lag Phenomenon.—One of the curious characteristics of the oculogravic illusion concerns the difference between the time required to subject a person to a change in direction of resultant force and the time required for the apparent displacement of the object in space as perceived by the subject. In studying this "lag" phenomenon,[3d] a procedure similar to that described above was used, except in the following particular: The subject was exposed to a predetermined level of centripetal force within three seconds and, three minutes later, decelerated to a stop within four seconds. A comparison was made of the time records of the change in

direction of resultant force and of the subject's rotation of the target line (Fig. 2). It may be remarked in passing that this "lag" is also observed with regard to the apparent change in body position.

It was found [3d] that the mean duration of the "lag" was 81 seconds in the case of three subjects exposed on 10 occasions each to centripetal forces where ϕ equaled 13.8 and 34.3 degrees. Initially the magnitude of the "lag" in degrees of arc equaled a large fraction of the angle ϕ, but it decreased rapidly, to become relatively small during the latter portion of the "lag" period. There was no definite evidence of overshoot. On deceleration, which required about four seconds, a similar, though smaller, "lag" was observed. Measurement of the "lag" was complicated by the fact that there was a tendency to overshoot.

COMMENT

The force of gravity is undoubtedly the most constant, and probably the most fundamental, influence to which man is subject. Its influence on his evolutionary

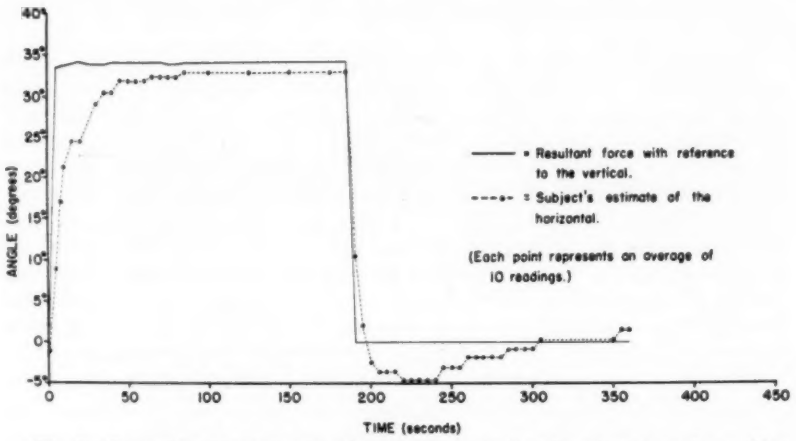

Fig. 2.—Comparison of actual and estimated changes in direction of resultant force. Note subject's delay in following time-force pattern.

development has been so great as to be well-nigh incalculable. It has dictated the need for, if not the means of, adaptation with regard to many physiological and psychological mechanisms. A coordinate system based on the direction of gravity forms the basis of a reference system by which man orients himself to the earth and to objects in space. In varying degree, most of the sensory organs aid in this orientation process, furnishing cues which may be classified as primary, or direct, and secondary, or indirect.

Direct cues are provided when certain sensory elements are stimulated by virtue of the fact that man has weight and rigidity and is in contact with the environment. Thus, deformation at points of contact and the relative displacement of bodily tissues set up tensions and pressures which are transmitted to all parts of the body and which constitute an adequate stimulus; an important example is the relative dis-

placement of the hairs in the sensory cells of the otolith organs. Orientation cues which originate in this manner are not only direct but inescapable; in a healthy person they are obligatory cues.

Indirect cues are provided through the perception of stimulus patterns, chiefly visual, which are presumed to be indicators of the reference system based on the direction of gravity. Thus, a plumb bob suspended by a string and a ruler at right angles to it furnish excellent visual cues which are not due to the direct influence of the force of gravity on the visual system. These indirect visual cues are abolished in darkness or when the eyes are closed and are, therefore, nonobligatory.

In the experiment described above, the subject was exposed to a change in direction of resultant force with reference to himself; it was as though the earth had changed its position and increased its mass. He perceived direct cues which tended to orient him to a new frame of reference, based on the direction of resultant force. The direct cues were in response to stimuli which were of greater intensity than normal. The subject also perceived indirect cues when viewing certain objects in the visual field. The amount of detail in the visual field was under the control of the experimenter, and objects always retained their original position with respect to the gravitational vertical. It was under these conditions of conflicting cues that the subject perceived the apparent change in position of objects in the visual field. It is worth while to speculate briefly on the possible cause of this phenomenon and its meaning in terms of human behavior.

There is evidence from the data presented that neither ocular nystagmus nor rotation of the eye can account for the oculogravic illusion. The apparent motion resulting from ocular nystagmus is characterized by rapid rate of movement without a corresponding degree of displacement of the object in space,[10] whereas in the case of the oculogravic illusion the correspondence is good. Furthermore, if a strong after-image is produced and superimposed on a real target in the visual field, the two will appear to separate if ocular nystagmus is induced, whereas they are displaced in the same direction under the conditions in the present experiment. Rotation of the eyeball cannot be the cause of the oculogravic illusion, for several reasons. First, the magnitude of the apparent displacement can exceed the range of eye-movements in certain directions; second, regardless of the meridional direction of apparent movement, the object always appears sharply defined, indicating foveal vision, and, third, an after-image moves in the "wrong" direction.

If the oculogravic illusion is not the result of some "reflex connection" at the level of the oculomotor nuclei, causing ocular movement, the origin must be sought in some psychophysiological mechanism which has no correlate in the retinal image. The visual phi phenomenon is one example; in this, the apparent movement is not related to the tracking of an image over the retina but is incidental to one's ability to perceive space.[11] Space is perceived through visual cues in the case of the phi phenomenon, whereas in the oculogravic illusion it is based on nonvisual cues, which directly orient us to resultant force. It is important to add, however, that the oculogravic illusion is not due simply to the fact that a person is aware of a

10. Patterson, J. L., Jr., and Graybiel, A.: Thresholds of Stimulation of Otolith Organ in Man as Determined by Oculogravic Illusion, to be published.

11. Verhoeff, F. H.: Phi Phenomenon and Anomalous Projection, Arch. Ophth. **24**:247-251, 1940.

change in body position and therefore alters his "projection" of the visual world. Experientially, we are aware that objects in the visual field may or may not shift in conformity with a change in body position. Even if the cause or origin of the oculogravic illusion is unknown, its meaning, in part at least, is clear. It is an example of "sensory cooperation" whereby visual cues are brought into conformity with cues which directly orient one to resultant force. Under certain conditions in our experiment this occurred despite all efforts to prevent it. Considering the low threshold for perception of the phenomenon, it is reasonable to suspect that under ordinary conditions, i. e., in the absence of exposure to centripetal acceleration, there is a similar tendency for cues which orient one to gravity to influence visual-space perception.

SUMMARY

When a person is subjected to a change in direction of resultant force relative to himself, he experiences an illusion wherein objects in the visual field, stationary with respect to the observer, appear to move and to assume a new position more or less in accord with the direction of resultant force. This phenomenon has been termed the oculogravic illusion.

Under the experimental conditions the subject perceived direct and indirect orientation cues. The former had their origin in stimulation of sensory elements by tension and pressures induced by resultant force. The indirect cues were provided by the perception of objects in the visual field which retained their original position with respect to the gravitational vertical. It was in the resolution of these conflicting cues that objects in the visual field appeared to move and to be displaced. The important variables determining the appearance of the illusion were (1) the pattern of centripetal acceleration, (2) the position of the subject, and (3) the visual framework.

When the direction of resultant force was shifted with respect to the subject, the illusion consisted of apparent movement and apparent displacement of objects in the visual field. If the relation between resultant force and subject was fixed, apparent displacement was observed, but not apparent movement.

Under favorable conditions the following characteristics of the illusion were observed:

(*a*) The threshold of perception was low.

(*b*) The apparent movement was in the nature of a smooth, gliding motion, and there was little or no overshoot.

(*c*) There was good correspondence between the rate of apparent movement and the magnitude of apparent displacement.

(*d*) The illusion behaved in a predictable manner, and there was a close correspondence between the estimated degree of displacement and the actual change in direction of resultant force.

(*e*) The apparent displacement showed little or no adaptation.

(*f*) If the change in direction of resultant force was abrupt, a "lag" was observed; i. e., the period required for the subject to perceive the change was much longer than the period during which the change in resultant force took place.

When a person was exposed to a change in direction of resultant force of considerable magnitude, the orderliness of the illusion was replaced by a more striking phenomenon wherein the response was no longer linear.

Reasons are given which indicate that the oculogravic illusion is not due to eye movement. Thus the cause must be sought in a psychophysiological integration that allows for the perception of movement which has no correlate in the retinal image. This may be incidental to our space-perception ability.

The oculogravic illusion is an example of sensory interaction whereby visual cues are brought into conformity with cues which orient us to resultant force. It is reasonable to suspect that this interaction functions, in some degree at least, under ordinary living conditions and influences our behavior.

The oculogravic illusion offers a means of studying cooperation and rivalry of certain sense departments.

The oculogravic illusion is experienced in flight and is important in aviation medicine.

FALSE SCOTOMAS ASSOCIATED WITH HIGH UNCORRECTED REFRACTIVE ERRORS

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A CAUSE of false scotoma is deserving of presentation because the potential frequency of the type of scotoma is greater than the combined incidence of all other types and because false scotoma simulates a genuine scotoma so closely that it may be misleading.

At the University of Michigan eye clinic we use a 10-mm. white circle for a fixation target at 1 meter on the black campimeter screen; and when searching for central scotomas we not infrequently move the appropriately sized object out from this central target until the patient says that he sees it. With this method, however, a central scotoma almost invariably appears under cycloplegia in patients with uncorrected ametropia. The intensity of this scotoma depends on the degree of ametropia and is essentially unchanged by changes in illumination, ranging from ordinary indoor lighting to very subdued light. If there is no associated macular or other pathologic change, this scotoma will always disappear completely when the patient looks through his correcting glasses. This finding may readily be duplicated by anyone examining a patient with high ametropia. The most probable explanation is that the blurred image of the central fixation target overlaps and obscures any smaller objects in its immediate vicinity, the extent of the blurring depending on the amount of ametropia and the size of the pupil.

That this is indeed the explanation of the "refractive scotoma" is readily proved by shifting fixation to a small black tack (or, if this is not visible, to a blank area midway between two white targets). Any object large enough to be seen anywhere in the field will be seen centrally immediately under these conditions regardless of how large a "scotoma" has been demonstrated to surround the 10-mm. white fixation target. (A subject with very high ametropia will not, of course, see a 1-mm. white object at 1,000 mm. anywhere—perhaps not even a 2- or 4-mm. object will be focused well enough at 1,000 mm. to be appreciated.) It has not been possible to demonstrate "refractive scotoma" in any patient (no matter how great his ametropia) when he is fixating the black pin or the blank area between two white targets. All scotomas demonstrated under these conditions are genuine and will be reflected in the impossibility of producing normal vision with correction.

Through the relations of similar triangles, one may calculate the approximate size of a blur circle for any given degree of ametropia. Table 1 (roughly calculated

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for the schematic eye) gives the distance, expressed in millimeters, that two objects must be apart in order to be perceived as completely separated when viewed at 1 meter with the given degree of ametropia. This distance increases in direct proportion to the diameter of the pupil, thereby explaining why the "refractive scotoma" is so conspicuous in mydriatic patients.

Table 2 is a similar tabulation of figures derived experimentally. The number given expresses the distance in millimeters (average of three readings for each)

TABLE 1.—*Theoretical Values for the Millimeters of Separation Necessary for Seeing Two Completely Separate Objects at 1 Meter*

	Pupillary Diameter, Mm.		
	1.5	3	6
Error, D.			
Myopia			
— 1.00	0	0	0
— 4.00	3	6	12
— 7.00	6	12	24
—10.00	10	20	40
—13.00	13.5	27	54
Error			
Hyperopia			
2.00	3	6	12
5.00	5	10	20
7.00	7.5	15	30
11.00	9.5	19	38

TABLE 2.—*Experimental Values for the Radius, in Millimeters, of the "Refractive Scotoma"*

	Pupillary Diameter, Mm.		
	1.5	3	6
Lens Simulating Myopia, D.			
4.00	0	4 mm. 1/1000	6 mm. 1/1000
7.00	9 mm. 1/1000 3 mm. 2/1000	20 mm. 1/1000 7 mm. 2/1000	25 mm. 1/1000 14 mm. 2/1000
10.00	7 mm. 2/1000 4 mm. 4/1000	20 mm. 2/1000 9 mm. 4/1000	18 mm. 2/1000 14 mm. 4/1000
13.00	17 mm. 2/1000 7 mm. 4/1000	28 mm. 4/1000 12 mm. 8/1000	20 mm. 4/1000 17 mm. 8/1000
Lens Simulating Hyperopia, D.			
— 2.00	0	3 mm. 1/1000	3 mm. 1/1000
— 5.00	2.5 mm. 1/1000	10 mm. 1/1000	9 mm. 1/1000 3 mm. 2/1000
— 8.00	4 mm. 1/1000	18 mm. 1/1000 7 mm. 2/1000	9 mm. 2/1000 5 mm. 4/1000
—11.00	10 mm. 2/1000 7 mm. 4/1000	11 mm. 2/1000 7 mm. 4/1000	15 mm. 2/1000 12 mm. 4/1000

between the edge of the white fixation target and the point at which the test object is first seen (margin of the scotoma). With hydroxyamphetamine (paredrine®) hydrobromide and homatropine cycloplegia and mydriasis, the subject observed the target through artificial pupils and lenses of the designated sizes and strengths. When values are not given for 1/1000 or 2/1000, the omission indicates that the subject was unable to see these objects anywhere in the field owing to the extremely blurred vision.

Tables 1 and 2 are strikingly identical in their progression toward a larger "refractive scotoma" with increasing ametropia and pupillary size. The considerable difference in absolute values is explained by the following facts:

1. The test object is perceived before its blur circle has completely separated from the blur circle of the fixation target. In fact, it seems from the patients' descriptions and the positions of the object that the test object is noticeable when these blur circles are only half separated, and form an elliptical blur. This phenomenon is more marked with the larger objects, which must be used with the higher degrees of ametropia.

2. The artificial pupil is situated about 20 mm. in front of the real pupil. If the values in Table 1 had been calculated on this basis, they would be uniformly reduced to about two-thirds their present magnitude. In other words, in order that a "refractive scotoma" may be duplicated experimentally, the blurring lens must be half again as strong as the ametropia of the patient whose scotoma one is duplicating.

An astigmatic patient would, of course, have an oval scotoma. For example (Table 1), — 13.00 sph. ⊃ + 6.00 cyl., ax. 90 with a pupil of 6-mm. diameter

TABLE 3.—*Clinical Values for the Radius, in Millimeters, of the "Refractive Scotoma"*

Patient	Refractive Error	Pupillary Diameter, Mm.	Radius of Scotoma
LA	— 1.50	..	None
TI	— 2.50 ⊃ 0.25 × 85	..	12 mm. 1/1000
HA	— 3.00 ⊃ 0.50 × 140	5	7 mm. 2/1000
HU	— 3.00 ⊃ 0.25 × 90	..	6 mm. 1/1000
LE	— 3.50 ⊃ 1.00 × 85	2.5	3-4 mm. 2/1000
HO	— 4.50 ⊃ 0.75 × 175	..	10 mm. 2/1000
GA	— 7.25	..	30 mm. 2/1000
LE	— 8.00 ⊃ 1.50 × 85	8	30 mm. 4/1000
JO	—16.50 ⊃ 0.75 × 148	6	20 mm. 8/1000
CO	+ 2.75 ⊃ 1.00 × 90	..	3-4 mm. 2/1000
NO	+ 5.50 ⊃ 1.62 × 82	3	15 mm. 2/1000

would give an oval scotoma extending 54 mm. from the central target in one meridian and 24 mm. in the other. Experimental dimensions of this scotoma are 20 by 12 mm. with 4/1000 white.

Table 3 presents sample scotomas actually observed in ametropic patients, tested without their glasses and using the 10-mm. white fixation target. These subjects were patients routinely passing through the visual-field room. None of these scotomas persisted when the patient fixed on a black pin through his glasses.

COMMENT

That the "refractive scotoma" is not an insignificant and rare phenomenon is obvious from the fact that it may be elicited in any patient with 3 D. or more of ametropia when under cycloplegia. Myopic, senile, or other types of degeneration often may seem to involve the macula seriously, and when the observation of such a lesion is coupled with the presence of a 2-degree ("refractive") scotoma for 2/1000 white the diagnosis of macular degeneration would seem evident—until one wonders why the patient can read 6/6 with correction! Recognition of the artifact of "refractive scotoma" will permit avoidance of such errors. I should like

to reiterate that a "refractive scotoma" is never present when the perimetrist uses a black fixation point and when the patient wears proper correcting lenses.

SUMMARY

1. A refractive error per se will produce a central "refractive scotoma" if this is plotted from a white fixation target.

2. The potential incidence of "refractive scotomas" is as great as the incidence of ametropia higher than 3 D.

3. "Refractive scotoma" is eliminated by having the patient wear correcting lenses and/or by utilizing a black pin for a fixation target.

RAEDER'S SYNDROME

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GEORGE Raeder,[1] a Norwegian neurologist, in 1918 first described a disturbance of the oculopupillary fibers associated with an incomplete Horner syndrome. The patient had headache, vomiting, pain in the left trigeminal area, paresis of the left side of the palate, and ptosis, miosis, and hypotonia, on the left, without vasomotor or trophic disturbances. The patient died of pulmonary tuberculosis, and necropsy revealed a tumor, described as an endothelioma, between the internal carotid artery and the Gasserian ganglion. The cervical sympathetic trunk was carefully examined down to the third thoracic segment without any lesion being found.

This case was of extreme interest, since it presented a sympathetic syndrome quite distinct in anatomical localization from the ordinary sympathetic syndrome produced by spinal lesions or lesions of the cervical sympathetic fibers. Raeder suggested, therefore, that lesions of the sympathetic fibers be classified as follows:

Spinal:	Incomplete Horner syndrome, often accompanied by other neurologic signs, such as Klumpke's paralysis, syringomyelia, or occlusion of posterior inferior cerebellar artery
Cervical:	Complete Horner syndrome, with as a rule, no other neurologic symptoms
Paratrigeminal:	Horner syndrome without vasomotor or trophic disturbances, generally accompanied by lesion of fifth cranial nerve

In 1924 Raeder reported four more cases of paralysis of the ocular sympathetic fibers,[2] with lesions localized to a limited space, which he believed justified the designation of paratrigeminal paralysis of the sympathetic fibers. A summary of the symptoms in these five cases is presented in the Table.

Peripheral lesions of the oculopupillary fibers have been described by others. Thus, Nieden,[2] in 1884, recorded a case of right-sided ptosis, miosis, enophthalmos, and anhydrosis of the forehead and eyelid in which he thought there was a lesion of the internal carotid plexus. Metzer and Woefflin,[2] in 1914 and 1916, described lesions of the sympathetic fibers in the carotid canal caused by suppurative otitis media. Bing,[2] in 1914, described paralysis of the sympathetic fibers due to herpes zoster, and Nagel,[2] in 1873, showed that hypotonia and miosis may accompany inflammation of the cornea. Cordes and Klauber,[2] in 1917 and 1918, described three cases of gunshot wound, with symptoms similar to those in Raeder's first

Presented at a meeting of the College of Physicians of Philadelphia, Section on Ophthalmology, April 17, 1952.

1. Raeder, G.: Et tilfalde av intrakraniel sympaticuslammelse, Norsk mag. lægevidensk. **79**:999-1015 (Sept.) 1918.

2. Raeder, J. G.: "Paratrigeminal" Paralysis of Oculo-Pupillary Sympathetic, Brain **47**:149-158 (May) 1924.

case. Finally, Sales, in 1939,[3] described a case of Raeder's paratrigeminal neuralgia with sixth nerve paralysis.

It may be worth while to review the distribution of the sympathetic fibers. The fibers originate in the ciliospinal center, between the sixth cervical and the fourth thoracic segment of the cord. The level of emergence of the various preganglionic fibers depends upon the functions they subserve, being from the eighth cervical to the sixth thoracic root (the pupillary fibers emerging from the eighth cervical to the third thoracic). The fibers pass upward to the superior cervical ganglion, where they synapse. From here, some of the postganglionic fibers accompany the external carotid artery to the skin of the face, while the majority of postganglionic fibers, which become intracranial, form two interlacing plexuses, the cavernous and the internal carotid plexus. The fibers to the pupil arise from the internal carotid plexus, run as fine filaments to the Gasserian ganglion, and join the ophthalmic division of the trigeminal nerve. They then pass forward via the nasociliary branch to the two long ciliary nerves and pierce the sclera close to the optic nerve,

Summary of Raeder's Cases of Paratrigeminal Syndrome

Case No.	General Symptoms	Cranial Nerves Affected	Etiologic Agent
1	Sympathetic symptoms, without vasomotor or trophic disturbances; homolateral pains in eye and head	V, III, IV, VI	Tumor in region of Gasserian ganglion
2	Sympathetic symptoms, without vasomotor or trophic disturbances; homolateral pains in eye and head	V, IV, VI	Unknown
3	Sympathetic symptoms, without vasomotor or trophic disturbances; homolateral pains in eye and head	V, II (paracentral scotoma)	Fractured base of skull (patient run over by motor car)
4	Sympathetic symptoms, without vasomotor or trophic disturbances; homolateral pains in eye and head	V	Unknown
5	Sympathetic symptoms, without vasomotor or trophic disturbances; homolateral pains in eye and head	V, II (optic nerve atrophy)	Probably fracture at base of skull (injured by run-away horse)

passing forward in the perichoroidal space, to innervate the dilator pupillae. Other fibers from the carotid plexus accompany the third nerve and supply the superior and inferior palpebral muscles, while a third group of fibers, according to Raeder, probably pass from the internal carotid plexus to the deep petrosal nerve and then to the sphenopalatine ganglion, to innervate the orbital muscle. The sympathetic fibers from both the cavernous and the internal carotid plexus which form the sympathetic root to the ciliary ganglion supply blood vessels (Fig. 1).

The clinical syndrome of Horner may result from a lesion at any place in the course of these fibers, but the completeness of the syndrome varies with the localization of the lesion. When the cervical trunk is injured (*D*, Fig. 1), all the symptoms are usually present. When the lesion involves the spinal cord, the symptoms are less complete but are often accompanied by other neurologic findings, as in syringomyelia (*E*, Fig. 1), or occlusion of the posterior inferior cerebellar artery (*F*, Fig. 1). If the lesion is intracranial, one may expect Horner's syndrome without trophic or vasomotor symptoms, but with cranial-nerve involvement (*A*, *B*, and *C*, Fig. 1).

3. Sales, M.: Raeder Paratrigeminal Syndrome (Horner Syndrome with Paralysis of the Sixth Cranial Nerves and Neuralgia of Ophthalmic Branch): Report of Case, Arq. Inst. Penido Burnier **5**:231-237 (Dec.) 1939.

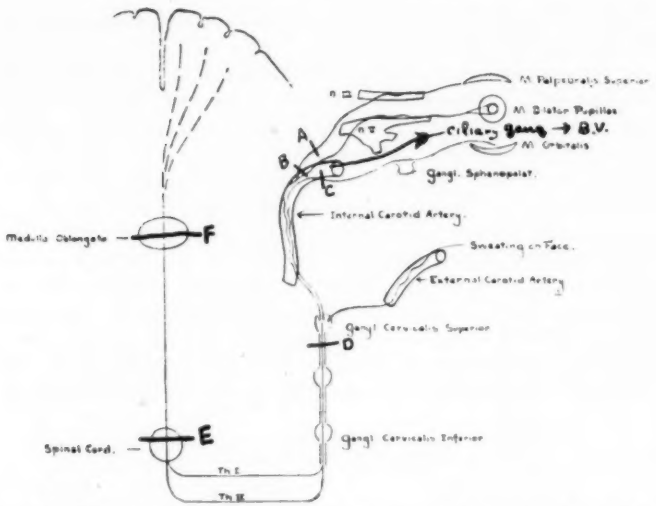


Fig. 1.—Distribution of the cervical sympathetic fibers. A lesion at *A* would produce ptosis; at *B*, ptosis and narrowing of the pupil; at *C*, theoretically enophthalmos; at *D*, all the symptoms of Horner's syndrome; at *E*, syringomyelia, and at *F*, occlusion of the posterior inferior cerebellar artery. Modified from Walsh, F. B.: Clinical Neuro-Ophthalmology, Baltimore, Williams & Wilkins Company, 1947, pp. 156-166.

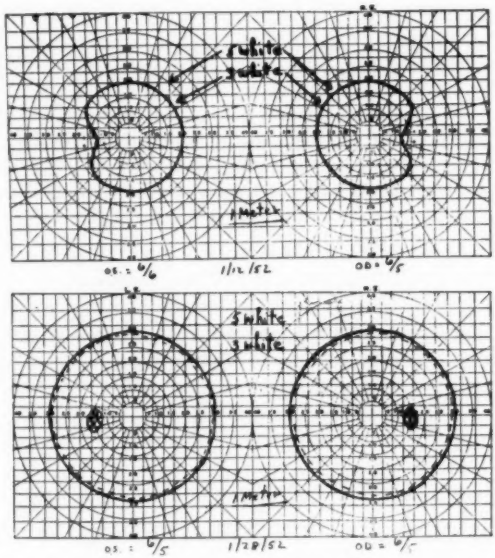


Fig. 2.—Perimetric determinations of the visual fields during the attack and after recovery, two weeks later.

REPORT OF A CASE

F. P., a man aged 59, was first seen on Jan. 9, 1952, at which time he complained of severe pain in the left eye and the left side of the forehead and drooping of the left lid, of about 10 days' duration. The pains seemed severer in the morning.

Ocular examination showed vision to be 6/5 in the right eye and 6/6 in the left eye. The extraocular muscles were normal. There were 2 mm. of ptosis and 2 mm. of miosis on the left side (right pupillary diameter, 5 mm.; left pupillary diameter, 3 mm.). Corneal sensation was diminished on the left, but there were no vasomotor changes or trophic disturbances on the left side of the face or forehead. The central fields showed bilateral general depression, with baring of the blind spots; but no central scotoma could be obtained in the blurred (left) eye. An attempt to compare the amplitude of accommodation of the eyes was unsatisfactory, owing to the blurred vision in the left eye. When 1 drop of 4% cocaine was instilled into each eye, the involved pupil remained unchanged, while the normal pupil dilated 3 mm. The fundus was normal, and tension measured 22 mm. in each eye.

The patient was told to return the next day for reexamination of the fields and an epinephrine test, but that day pain and swelling developed in the left cheek and the left side of the upper jaw. His dentist removed an abscessed left upper lateral incisor. The next day, when he was again seen by his ophthalmologist, he stated that his headaches and drooping lid were much better. His pupils were then equal in size; but when 1:1,000 epinephrine hydrochloride solution was instilled the normal pupil did not respond, whereas the previously miotic (left) pupil dilated 2 mm. Since the patient seemed to be improving, he was instructed to return one week later.

On Jan. 21, 1952, 12 days after the patient was first seen, he was again examined and found to have recovered normal vision, visual fields, corneal sensation, and pupillary reactions. He no longer had any headache and showed only a very slight ptosis, which had completely disappeared the following week.

Summary.—A white man aged 59 presented a left-sided Horner syndrome without vasomotor or trophic disturbances. The left pupil was refractory to cocaine and sensitive to epinephrine. He had blurred vision on the left, pain in the left trigeminal nerve, and diminished corneal sensation on the left. All these signs and symptoms pointed to intracranial involvement of the sympathetic fibers, such as is seen in Raeder's paratrigeminal syndrome. The interesting thing about this case is the complete recovery following extraction of an abscessed tooth. Focal infection therefore, should always be considered a possible cause in cases of Raeder's syndrome.

2027 Spruce St.

CORRECTION OF SENILE ENTROPION

Modification of Technique

SIDNEY A. FOX, M.D.
NEW YORK

APPROXIMATELY a year ago a new operation for the relief of senile entropion was reported.[1] It had been used in 14 lid operations, with no recurrences. The purpose of the present, short paper is (1) to report further on these cases, (2) to add five cases to the total, and (3) to submit a modification of the original technique.

Of the 10 patients with 14 lid operations reported in detail a year ago, 8 (11 lids) have been under observation throughout the past year. There have been no recurrences. Since the report an additional five lids (in four patients) with senile entropion have been operated on with the technique previously reported. Three patients are doing well; one, with bilateral entropion, had a recurrence.

Four weeks after operation there was a recurrence of the entropion in the third recent case, in which both lids had been repaired at the same time. This occurred in a patient who had abnormal "bagginess" of the lower lids, and it seemed likely that not enough skin-muscle tissue had been excised. An additional skin-muscle spindle, measuring 15 by 10 mm. at its widest point, was therefore resected on each side, and at the time of writing (seven months later) both lids are in excellent position.

As a result of this recurrence a modification of the operation has been worked out which, it is believed, will overcome the tendency. In the past five months five additional lids have been repaired with this modified procedure. All show excellent results. However, not enough time has elapsed to permit evaluation of the long-term efficacy of the method, although the advantages are obvious.

MODIFICATION OF TECHNIQUE

A spindle is marked out just lateral to the external canthus. This is 15 mm. in length and 8 to 12 mm. in width, depending on the laxity of the skin-muscle tissues. Its upper end is about 5 mm. above the external canthus. The entropic lid is then everted, and a triangle of tarsoconjunctiva is marked out at the center of the lid. This measures 5 mm. at the base, and its apex lies at the lid margin (Figure, *A*). The tarsoconjunctival triangle is resected, as is the skin spindle and all the included orbicularis fibers below the raphe (Figure, *B*).

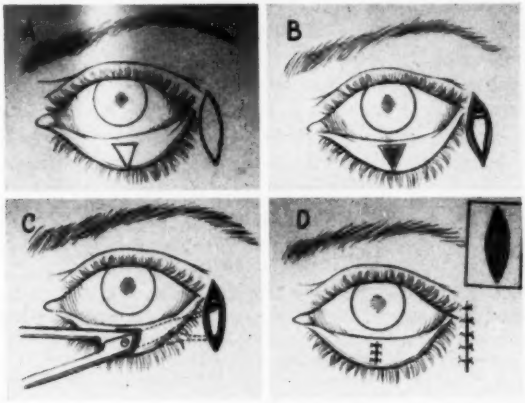
From the Eye Service, Goldwater Memorial Hospital.

1. Fox, S. A.: Relief of Senile Entropion, A. M. A. Arch. Ophth. **46**:424, 1951.

The temporal edge of the tarsal wound is lifted, and the skin-muscle layer is separated from the tarsoconjunctiva laterally with scissors until the scissor points appear in the bared skin-muscle opening. This dissection is carried to the lid margin, above (until it is seen to blanch), and to the lowest point of the skin-muscle spindle, below (Figure, *C*). Thus, enough mobility between skin-muscle and tarsoconjunctiva is created to obviate any skin puckers on closure of the wound.

The tarsal wound is closed with 4-0 braided black nonabsorbable (silk) surgical sutures U. S. P. The skin-muscle wound is closed with 5-0 braided black nonabsorbable surgical sutures U. S. P., care being taken to catch up the ends of the cut muscle in the sutures. Or the muscle wound may be closed separately with 3-0 plain absorbable surgical (gut) sutures U. S. P. (inset, Figure, *D*).

There is nothing in this modification which alters the basic principles of the previous technique, i. e., tarsus and skin-muscle are still resected to strengthen the



(*A*) A tarsoconjunctival triangle is marked out with its apex at the lid margin. The base measures 5 mm. A skin-muscle spindle just lateral to the external canthus is also marked out. This measures 15 mm. in length and 8 to 12 mm. at its widest point. Its upper point is 5 mm. above the external canthus.

(*B*) The tarsoconjunctival triangle is resected. The skin-muscle spindle is resected except for the muscle above the raphe.

(*C*) The skin-muscle tissue and tarsoconjunctiva between the two wounds are separated to permit closure without wrinkling of the skin.

(*D*) The tarsal wound is closed with 4-0 braided black nonabsorbable surgical (silk) sutures U. S. P. The skin wound is closed with 5-0 braided black nonabsorbable surgical sutures, care being taken to include the muscle also.

lid wall, as before. The only difference is that lid splitting is avoided and the position of the skin-muscle spindle is changed. The advantages are obvious:

1. Less surgery means a simpler technique and quicker, smoother healing.

2. Raising the position of the spindle increases the amount of skin-muscle excised at the level of the lid margin; hence there is a greater stiffening of the lid wall at the margin where it is needed most when the wound is closed.

The recurrence in the case reported emphasizes the importance of resecting enough skin-muscle tissue, as well as tarsus, to strengthen the lid wall. Since laxity of lower-lid tissues varies with different persons, the amount of skin-muscle excised must also be varied to suit the case.

SUMMARY

A simplified modification of the technique originally reported for the repair of senile entropion is submitted. In the past 2½ years 19 lids have been operated on, with recurrence in one case of bilateral entropion. This was repaired by the resection of more skin-muscle tissue. In the last six months repair of senile entropion has been done in five additional cases according to the modified technique, with no recurrences. It is much too early for evaluation of results.

OPTIC NERVE PRESSURE BY ANEURYSM RELIEVED BY DECOMPRESSION OF OPTIC NERVE

Report of a Case

MAURICE J. HAUSER, M.D.
AND
HARVEY GASS, M.D.
DETROIT

INTRACRANIAL surgery in the treatment of aneurysms of the circle of Willis is rapidly gaining a foothold in many clinics. When the aneurysm impairs the function of the oculomotor nerve, the ophthalmic division of the fifth nerve or, especially, the optic pathways, symptoms are produced which frequently bring the patient to the ophthalmologist. In the case to be reported rapidly developing blindness was arrested and corrected by intracranial treatment of an aneurysm arising at the junction of the internal carotid and the ophthalmic artery. In this case, although no definitive treatment of the aneurysm itself was possible without great risk to the patient's vision, pressure on the optic nerve by the aneurysm was relieved by decompression of the nerve in the optic foramen.

REPORT OF CASE

A white woman aged 34, a housewife, was first seen on March 22, 1950, because of blurred vision in the right eye and an associated supraorbital dull, throbbing right-sided headache, of 10 days' duration. She stated that although the left eye had been turned inward and almost blind since childhood, she had always had good visual function in the other eye. Her past and systemic histories were not contributory.

General inspection of the patient neither revealed nor suggested an acute illness. External examination of the eyes failed to reveal any abnormality except left concomitant esotropia of 15 degrees, which increased to 25 degrees for near. Visual acuity was 20/50 in the right eye, not correctible, and there was loss of the right superior peripheral visual field as shown in Figure 1 *A*. Visual acuity in the left eye was barely 20/400, and visual fixation in this eye could not be maintained. With a 5-mm. white target at 330 mm. and the patient's finger as a fixation point, one could not demonstrate any peripheral visual field defect in the left eye. The corneal sensitivity and anterior segment of each eye were normal, as were the pupillary reactions and tactile tension. Light finger pressure over the ciliary body in the right eye at 10 o'clock produced an aching type of pain; yet no cells were present within the right anterior chamber. Both ocular fundi were visualized well. In the right eye the disk margin was indistinct, with vessel parallax demonstrable above and below. The small central physiological excavation of the disk was retained. The retinal veins were somewhat dilated, presenting an artery-vein ratio of 1:3; but there were no hemorrhages or exudates. These findings of blurred disk margin, vessel parallax, retained central physiological excavation, and full retinal veins were interpreted as papilledema, of 1 D. The optic nerve head, retinal vessels, and macula were normal in the left eye.

A detailed neurological examination and general physical examination revealed nothing abnormal. Visual examination the following day, March 23, demonstrated a further loss in her right peripheral visual field (Fig. 1 *B*).

On March 24, she was admitted to the Alexander Blain Hospital. At that time central visual acuity in her right eye was still 20/50; yet the right field of vision was reduced to an inferior island, as shown in Figure 1 *C*. The right fundus was unchanged, and the left optic nerve head was still normal.

The findings in x-ray studies of the skull were considered normal. The pineal gland was not visualized; the sella turcica was well calcified and showed no erosion of the clinoid processes. The tuberculum sellae was not thickened. Special views of the optic foramen showed that

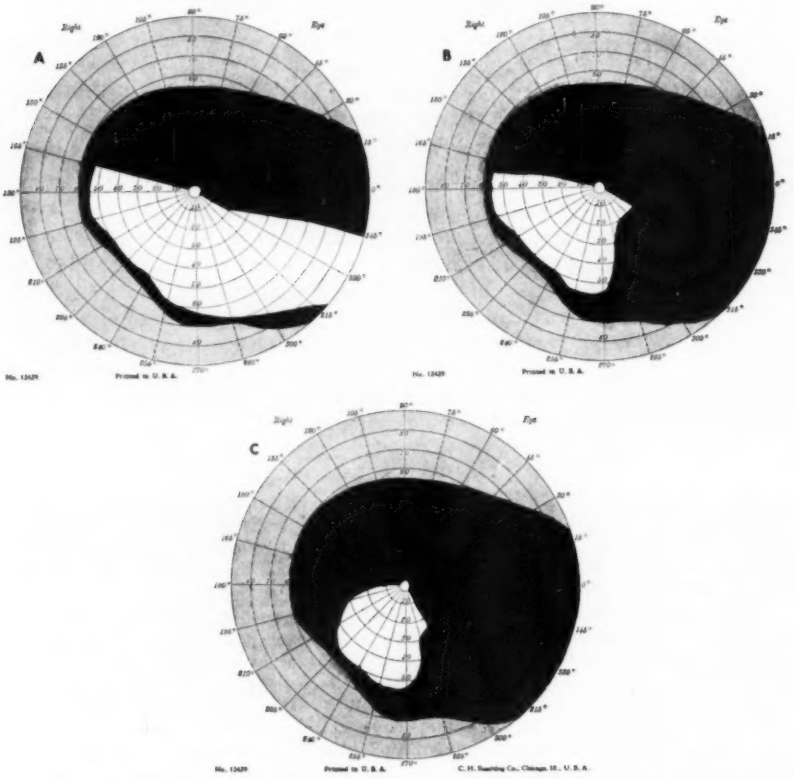


Fig. 1.—*A*, loss of the peripheral visual field for 3/330 white in the right eye (March 22, 1950). *B*, further loss of the right peripheral visual field (March 23). *C*, reduction of the peripheral visual field in the right eye to an inferior island (March 23).

the right foramen was slightly larger than the left, but without definite erosion, although the inferior segment of the optic foramen was not as well calcified as the remainder. The serologic reactions of the blood were negative. Urinalysis and complete blood count gave results within normal limits. A right percutaneous cerebral arteriogram was done; there was no displacement of the anterior or middle cerebral arterial system or any abnormality in the region of the circle of Willis, in particular no aneurysmal lesion could be detected. A pneumoencephalogram failed to show any deformity or displacement of the ventricular system, and the basal cisterns were adequately filled.

By the following morning, March 25, the right eye had lost all light perception. The previously crossed and ambl opic left eye was doing a good job of fixation, while her right eye was now turned inward. Because she was rapidly going blind, it was decided to explore the parasellar region in search of a possible prechiasmatic lesion, not revealed by arteriogram or pneumoencephalogram.

She was transferred to Grace Hospital, Northwestern Branch, on March 26, 1950. The following day, with the use of intratracheal anesthesia, a right frontal osteoplastic craniotomy was performed through a coronal incision in the scalp. A dural flap was reflected, with its base toward the midline, and the right frontal lobe was then retracted, exposing the chiasmal area. The left and right optic nerves and the optic chiasm were seen easily, and no specific lesion was noted. The internal carotid artery, coursing laterally from beneath the right optic nerve, was identified. No evidence of aneurysm could be seen arising from this visible portion of the internal carotid artery. The right optic foramen was then unroofed for a distance of about 8 mm., until the beginning of the orbit was identified. With gentle traction exerted with a blunt nerve hook, the optic nerve was retracted medially, exposing the internal carotid

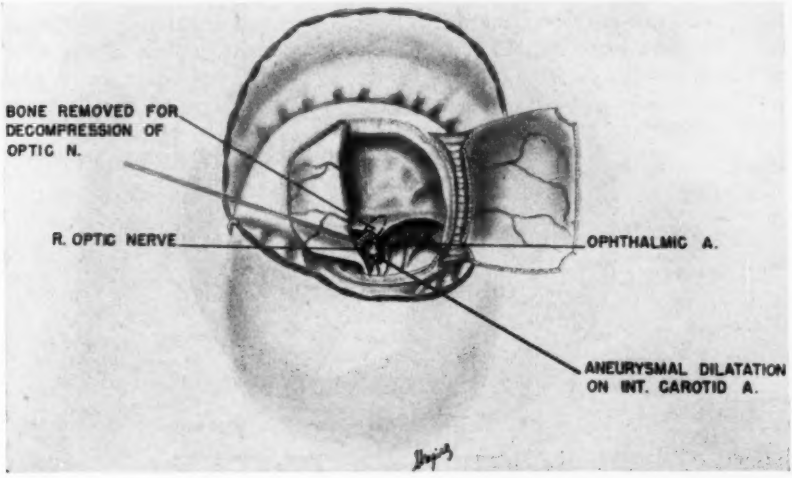


Fig. 2.—Exposure of dilatation at the junction of the ophthalmic and internal carotid artery with pressure on the right optic nerve.

artery proximally toward the optic foramen. As this was done, there came into view a small aneurysmal dilatation, about the size of half a pea, at the junction of the ophthalmic and the internal carotid artery (Fig. 2). This dilatation obviously had formed a knuckle which had pressed upward against the optic nerve. The dilatation had no neck, so that it could not be clipped; nor was it considered feasible to trap the aneurysm with clips on the carotid artery on either side of the aneurysm, since doing so not only would sacrifice the right internal carotid artery but also would deprive that eye of its blood supply through the ophthalmic artery on that side. It was decided, therefore, to extend the decompression of the optic nerve and to provide at least palliative relief of her visual loss. The bony decompression area was then enlarged even farther, for a total distance of about 12 mm., until the optic nerve was seen to ride up over the aneurysmal dilatation in a gentle curve, without pressure. After hemostasis was gained, the wound was closed in the usual fashion.

The patient tolerated the procedure well and was ambulatory within three days. The remainder of her postoperative course was uneventful. Immediately after the operation, vision in the right eye improved to recognition of large forms, and day by day she showed rapid recovery of visual function. After one month the edema of the right optic nerve had subsided,

and visual acuity had improved to 20/30, with restoration, of all but the right inferior nasal field of vision (Fig. 3). Two years after operation she was asymptomatic and was working at an inspection job in a factory, with an unaided visual acuity of 20/25 in the right eye. Repeated visual-field studies continue to reveal a defect of the right inferior nasal quadrant, which causes no subjective symptoms.

COMMENT

The rapid development of optic nerve pressure in the patient's remaining useful eye created a situation of great urgency. The decision to perform an exploratory operation in the face of a normal pneumoencephalogram and a normal cerebral arteriogram (in retrospect, a sight irregularity in the carotid siphon at the approximate location of the aneurysm could be seen in the arteriogram, but was not sufficient to justify pathological interpretation) was made on the basis of unilateral papilledema and progressive loss of the homolateral peripheral visual field,

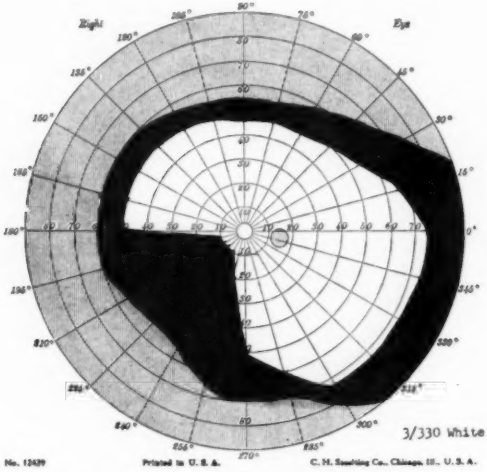


Fig. 3.—Restoration of the right peripheral visual field for 3/330 white except for the inferior nasal quadrant, one month after decompression of the optic nerve.

suggesting a prechiasmal lesion. The prospect of imminent blindness made the decision easier. Even though nothing pathological was evident on complete visualization of the optic nerve and the visible portion of the internal carotid artery, exploration of the optic nerve into the orbit was deemed necessary to exclude absolutely any pressure against this nerve. In carrying out this procedure, by mobilizing the optic nerve through decompression of the optic foramen, we discovered the aneurysmal lesion.

One can only speculate as to the incidence of similar lesions which go undetected in less urgent situations. How long this gratifying return of function will persist is, of course, uncertain. Undoubtedly, this aneurysmal dilatation was congenital, but it ultimately expanded sufficiently to compromise the overlying optic nerve. Should loss of visual field recur in that eye, we shall probably have to assume that the aneurysm has once more expanded, and it is possible that further decompression may again be of value.

The loss and return of visual field deserve special mention. The first impairment occurred above (Fig. 1*A*), and within a few days only an inferior island of peripheral vision remained (Fig. 1*C*). If one can assume that all peripheral fibers within the optic nerve are equally vulnerable, any permanent impairment of peripheral vision should be at the site of the initial loss, that is, superiorly. Figure 3 demonstrates the reverse to be true. In explanation of this paradoxical field, one might suggest that the relatively soft aneurysm did not cause any permanent pressure damage to the optic nerve, but that the displacement of this nerve against the bony roof of the optic foramen did result in some pressure degeneration of nerve fibers. Moreover, one cannot exclude the possibility of surgical trauma to the nerve at the time of its decompression.

SUMMARY

A case of rapidly developing blindness in the only useful eye of a woman aged 34 is presented. Despite a normal cerebral arteriogram and normal pneumoencephalogram, on the basis of unilateral papilledema and progressive peripheral visual loss in that, her only useful, eye, intracranial exploration of the optic nerve was carried out. This revealed an aneurysm at the junction of the ophthalmic and the internal carotid artery, which pressed up against the overlying optic nerve. This was treated by liberal decompression of the optic nerve at the optic foramen, with a gratifying, sustained return of visual function to normal.

7411 3d Ave. (Dr. Hauser).

74 W. Adams St. (Dr. Gass).

Clinical Notes, New Instruments and Techniques

NEW QUADRIFOCAL SPECTACLE FOR THE PRESBYOPIC OPHTHALMOLOGIST

CONRAD BERENS, M.D.
NEW YORK

ABOUT 1940, it seemed desirable to have a single pair of glasses that would facilitate specific visual tasks without the necessity of wearing a loupe [1] or changing glasses. Mr. Conrad Carlsen was asked to construct lenses that would fill the specified requirements. The spectacles are described now because of the interest shown in them by many ophthalmologists during the last 12 years.

The requirements were spectacles that would facilitate the following visual activities:

1. Permit distance vision without the necessity of bending the head too far forward.

2. Permit clear intermediate vision for tasks at about 20 in. (50 cm.), for example, reading the markings on trial frames and for picking up instruments in operating.

3. Permit reading at a distance of approximately 16 in. (40 cm.), a distance which also would be satisfactory for plastic surgery and muscle operations.

4. Permit clear vision and little strain on convergence when dilating puncta, removing cilia, and performing corneal transplantations and cataract extractions, at an approximate distance of 10 in. (25 cm.).

Straight-top trifocal lenses which are supplied with the intermediate section (upper part of the segment), one-half the total reading addition, were selected. Below the reading section was cemented a segment of the desired focus which had incorporated with it the required prism to minimize strain in convergence.

These three working areas were inset from the interpupillary distance, so that the eyes would center in each of these three segments. A 3-mm. convergence for the normal reading distance was arranged for. Through the fourth section (because of increased convergence), it was found necessary to inset the segment 1 mm. in addition to the inset for reading purposes (Fig.).

The first spectacle had no prism in the segments, but prolonged work at approximately 10 in. produced eyestrain and headache after several hours. For this reason, a 1 Δ prism was first inserted base in before each eye, and later it was found more satisfactory to incorporate a 2 Δ prism base in each lens.

This study was aided by a grant from the Ophthalmological Foundation, Inc.

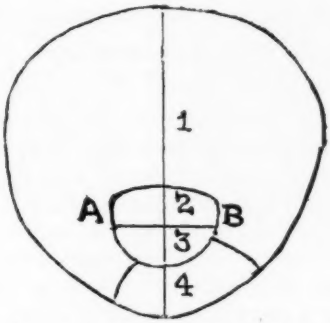
The spectacles were made by C. P. Carlsen, of A. Haustetter, Inc., New York.

From the Department of Ophthalmic Research, New York Eye and Ear Infirmary, and the Department of Ophthalmology, the Post-Graduate Medical School of New York University-Bellevue Medical Center.

1. Berens, C., and Losey, R. R.: A New Arrangement of the Beebe Loupe, Tr. Sect. Ophth., A. M. A., 1928, p. 417.

The last pair of quadrifocal spectacles was made from the following prescription:

O. D.: +2.25 sph. ⊃ —1.00 cyl., × 20 1 △ prism base down
O. S.: +2.00 sph. ⊃ —1.37 cyl., ×171 1 △ prism base up
+1.25 sph. add O.U. for intermediate
+2.50 sph. add O.U. for reading
+4.50 sph. and 2 △ prism base in O.U. for fourth, or operating, segment



A new quadrifocal spectacle for the presbyopic ophthalmologist: (1) distance; (2) intermediate, 6 mm.; (3) reading, 10 mm.; (4) operating, 9 mm. Width of segment (*A-B*), 22 mm.

The quadrifocal spectacles have proved especially useful for the numerous visual tasks demanded of the ophthalmologist. The upper part of the segment permits distance vision; the intermediate segment affords clear vision at about 20 in.; the normal reading segment is excellent for tasks performed at approximately 16 in., and the lowest segment permits clear vision for such delicate surgical procedures as corneal transplantations and cataract extractions at a distance of about 10 in.

708 Park Ave.

CORNEAL TRANSPLANTATION

Special Reference to Plastic Moist Chamber for Donor Eye

CONRAD BERENS, M.D.
NEW YORK

IN ORDER that a sterile, moist donor cornea may be assured in corneal transplantations, a two-section plastic moist chamber with a top has been devised.[1]

The base of the moist chamber (Fig., *A*) is lined with sterile isotonic sodium chloride-soaked gauze and is deep enough to hold the donor eye.

A graft of suitable size and thickness is excised from the cornea after first outlining the disk with a trephine. In lamellar keratoplasty, a special knife [2] is inserted to the desired corneal depth and, with a gentle, firm sawing motion, the graft is freed to the upper extremity of the outlined disk, then to the lower edge. Before the graft is completely excised, two double-armed nonabsorbable surgical (silk) sutures are inserted through the anterior edge of the corneal disk at the opposite extremities of the horizontal meridian. The graft may also be dissected with the Paufique and Desmarres knives and completed with corneal scissors. The disk may be placed in special clover leaf forceps [3] for completion of suturing, or a corneal suturing spatula may be applied to the cornea as the two other sutures are inserted at the ends of the vertical meridian.

In complete keratoplasty the trephine perforates half the cornea. Two sutures are introduced in the horizontal meridian and brought through the perforated plunger of a special trephine.[4] The cornea is then perforated, and the graft is excised with scissors if perforation is incomplete. The graft is placed in the clover leaf forceps, and two more double-armed sutures are placed at the ends of the vertical meridian.

The donor graft is carefully trimmed with corneal scissors and placed on the donor eye in the special moist chamber. The sutures are brought through the four slits in the rim of the moist chamber (Fig., *B*). In this manner the sutures are kept neatly in place and free from entanglement.

This study was aided by a grant from the Ophthalmological Foundation, Inc.

From the Department of Ophthalmic Research, New York Eye and Ear Infirmary and the Department of Ophthalmology, The Post-Graduate Medical School of New York University-Bellevue Medical Center.

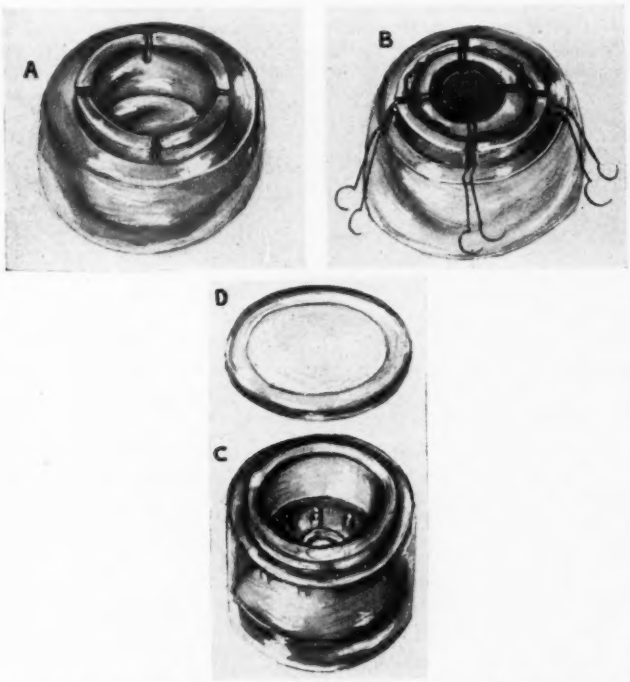
1. Made by R. O. Gulden, Philadelphia.

2. Berens, C.: Knife for Excision of Corneal Scars and for Bullous Keratitis, Tr. Sect. Ophth., A. M. A., 1940, p. 290.

3. Berens, C.: Special Suturing and Fixation Forceps, A. M. A. Arch. Ophth. **48**:348, 1952.

4. Made by Storz Instrument Company, St. Louis.

The top section of the plastic moist chamber (Fig., *C*) is provided with a lip which fits over the base snugly and has a perforated dome, which allows circulation of air. By insertion of additional saline-soaked gauze into the upper part of the chamber, the donor graft is prevented from drying while the scarred graft is being removed from the patient's eye.



Plastic moist chamber. *A*, base of moist chamber with suture slits in lip; *B*, excised and sutured donor graft replaced on donor eye in base of moist chamber with sterile gauze moistened in isotonic sodium chloride solution. *C*, top-domed and perforated section, allowing circulation of air; *D*, cover of moist chamber.

The cover (Fig., *D*) is applied until the donor graft is ready for use.

The special plastic moist chamber is light in weight and is sterilized in the manner usual for plastics.

Use of the plastic moist chamber with slits in the base lip avoids tangled sutures and facilitates the application of a sterile, moist donor graft to the recipient eye.

708 Park Ave.

Registry of Interesting Cases

It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.

BILATERAL SYMMETRICAL DYSTROPHY OF BOWMAN'S MEMBRANE

Report of a Case

THE DYSTROPHIES of the cornea and their classification is still a moot subject. In 1950 Franceschetti and Forni [1] compiled a chronological enumeration of 17 classifications of the heredofamilial degenerations of the cornea. The number of these classifications may be explained by the fact that they were formulated as new cases were added to the literature. In 1940 Franceschetti and Streiff [2] gave a systematic review of the various forms of hereditary dystrophies of the cornea. Histopathological studies made possible by keratoplasty were reported by Franceschetti and Babel [3] in 1950.

The present case, one of bilateral symmetrical dystrophy of Bowman's membrane, does not fall into any of the suggested categories, and the literature does not contain a case with similar characteristics.

CASE REPORT

L. R. E., a 19-year-old Neapolitan girl, came to the clinic for a pair of glasses. She had a florid complexion but was otherwise physically normal. There was nothing noteworthy in her history and no known ocular disease in her family. The right eye could not be examined with the ophthalmometer because of the irregular astigmatism. Examination of the left eye was difficult.

With ordinary illumination no abnormality of the cornea could be seen. Lateral illumination revealed a very slight, symmetrical opacity that ran vertically 2 to 3 mm. from the pupillary zone. Examined with the diffuse illumination of the slit lamp, this opacity resembled a thin china-white zone. On the temporal side its edge was clear and broken and could be compared to the outline of delicate lace; on the nasal side it gradually decreased, occupying the medial third of the cornea as far as the limbus. The pupillary portion of the opacity contained many clear lacunae. Parallel to the inferior half of the broken edge of the opacity, and 1 to 2 mm. from it in the transparent zone of the cornea, was a thin brown line. In the optical section of the slit-

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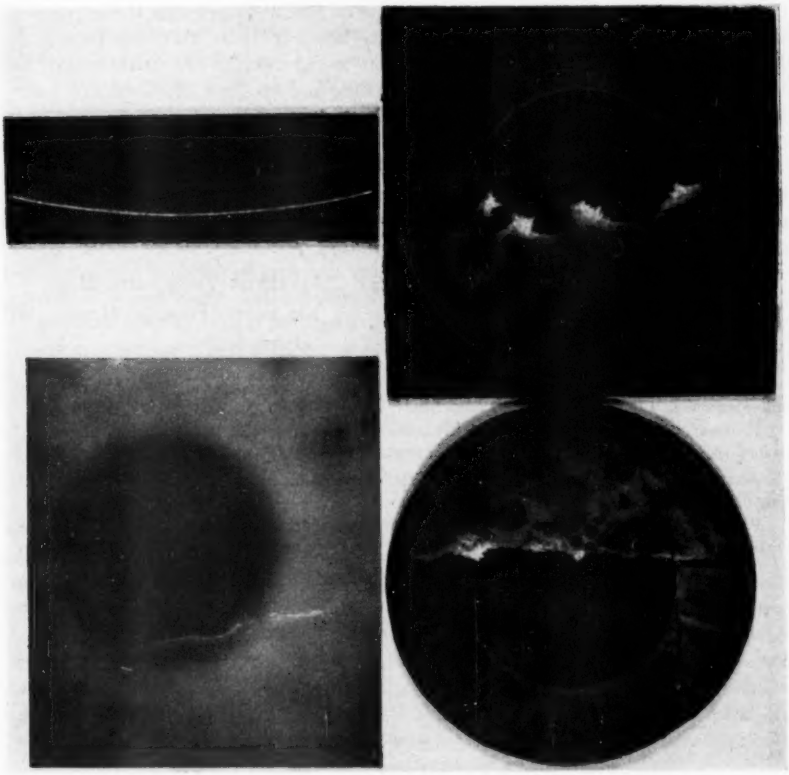
1. Franceschetti, A., and Forni, S.: II. The Heredofamilial Degenerations of the Cornea: A. Clinical Aspects, Acta XVI Concil. Ophth. (Britannia) **1**:193-244, 1950.

2. Franceschetti, A., and Streiff, E. B.: Hereditary and Constitutional Dystrophies of the Cornea, in Modern Trends in Ophthalmology, edited by F. Ridley and A. Sorsby, London, Butterworth Co., Ltd., 1940, pp. 414-427.

3. Franceschetti, A., and Babel, J.: II. The Heredofamilial Degenerations of the Cornea: B. Pathological Anatomy, Acta XVI Concil. Ophth. (Britannia) **1**:245-266, 1950.

lamp beam the opacity was seen to be localized at the level of Bowman's membrane. The more superficial parts of the adjacent parenchyma were perhaps involved. Measured with the Puglisi-Durante esthesiometer,[4] the sensibility of the cornea was found to be normal. The intraocular membranes were normal. Examination after the instillation of fluorescein showed the corneal epithelium to be intact. Laboratory tests for venereal infection and tuberculosis gave negative results.

These clinical findings led to the diagnosis of dystrophy involving Bowman's membrane.



Drawings of the cornea: upper left, right eye; upper right, left eye (diameters 4.5 cm.); lower left, optical section, 4 by 1.5 cm.; lower right, photograph, 4.5 by 4.5 cm.

Vogt [5] reported two cases of emmetropic, normal young persons who had small, homogeneous-looking spots at the level of Bowman's membrane. In these cases fluorescein tests showed the corneal epithelium to be intact. In the familial cases

4. Puglisi-Duranti, G.: Sensibilità fisiologica della cornea umana; nuovo estesiometro corneale, Arch. fisiol. **33**:359-372, 1934.

5. Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, Berlin, Springer Verlag, 1930, Vol. 1, p. 45.

reported by de Schweinitz and Cowan,[6] Berardi and Motolese,[7] and Bucklers [8] specific degeneration of Bowman's membrane was coupled with impairment of the corneal epithelium.

The dystrophy in the present case was bilateral and symmetrical and involved Bowman's membrane. In addition, there was in each eye a pigmented line parallel to, and 1 to 2 mm. from, the opacity, thus lying in the transparent zone of the cornea. Slit-lamp examination of this pigmented line showed it to be at the level of Bowman's membrane. It was like the line of Stähli, except that it was vertical instead of horizontal. It is suggested that the line was the result of the rupture of Bowman's membrane in the normal portion of the cornea. The thick opacity of the dystrophic area may be due to the shrinking and breaking of Bowman's membrane. This hypothesis is confirmed by the histopathological studies of Vogt [5] on the line of Stähli.

The present case, and perhaps Koby's [9] case, must therefore be included with the unclassified forms of hereditary dystrophy of Bowman's membrane. Future examinations in similar cases will provide a fuller explanation of this type of dystrophy and aid in its eventual proper classification.

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6. de Schweinitz, G. E., and Cowan, A.: An Unusual Corneal Lesion, Probably Congenital and Familial in Character: A Slit-Lamp Study, Am. J. Ophth. **16**:7-11 (Jan.) 1933.

7. Berardi, M., and Motolese, A.: Distrofia corneale ereditaria familiare con disepitelizzazioni recidivante, Boll. ocul. **17**:711-728 (Sept.) 1938.

8. Bucklers, M.: Über eine weitere familiäre Hornhautdystrophie (Reis), Klin. Monatsbl. Augenh. **114**:386-397, 1949.

9. Koby, F. E.: Sur la dégénérescence réticulaire superficielle de la cornée, Arch. opht. **44**:149-166 (March) 1927.

Annual Reviews

NEURO-OPHTHALMOLOGY

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DURING the past year the number of published papers dealing with some phase of neuro-ophthalmology increased over that of preceding years. This is evidence neither of newly aroused interest in the field nor of any remarkable progress; it is merely that more people are writing papers. In order that this review may be kept within prescribed limits, a number of reports of cases and articles of minor interest will be omitted.

PERIMETRY

Chamlin and Davidoff [1] emphasize that in the study of visual fields it is not enough to indicate a loss for one particular test object. In order that the depth of the loss in the defective region and the quality of the preserved field may be determined, it is necessary that a series of graded test objects be employed. Unless targets are selected with a particular purpose in mind, much time may be wasted. These authors found it convenient to divide the field into three regions: central (0 to 10 degrees), intermediate (10 to 25 degrees), and peripheral (25 to 90 degrees). For testing the peripheral portion of the field, they prefer the 2/330 white target; for the intermediate and central fields the 1/2000 target is used. When a visual defect is present, its intensity must be designated by use of targets as large as 100/330 or 40/330 if necessary. Once the quality of the defective field is ascertained, the better portion of the field is explored with small stimuli until the minimal stimulus is found. Several specific visual disorders are described, and a selection of test objects is recommended for each. The authors use a 2/2000 white target for measurement of the physiologic blind spot and for the detection of arcuate defects in glaucoma. They advise a 5/2000 red target for the discovery of minimal changes in retrobulbar neuritis. For suprageniculate lesions, they have found that the defect shows up best with 1/330 and 2/330 white targets. This reviewer is in hearty accord with their judicious selection of minimal test targets. These are certainly ideal for patients who are alert and cooperative. However, many patients who come for study of the perimetric fields are elderly or ill and their cerebral function is impaired. For them, slightly larger targets may elicit more reliable responses.

Study of defects in the visual fields usually provides reliable information. Klingler and Condrau [2] call attention to certain exceptions, noting that defects in the fields of vision that occur late in the course of brain tumor cannot be used for purposes of localization. Distant effects are particularly frequent in cases of internal hydrocephalus caused by cerebral tumor. In their opinion, central scotoma is caused by pressure of the optic nerve against the bony optic canal or by vascular disturbance.

1. Chamlin, M., and Davidoff, L. M.: Choice of Test Objects in Visual Field Studies, Am. J. Ophth. **35**:381-393 (March) 1952.

2. Klingler, M., and Condrau, G.: Lokalisatorisch irreführende Gesichtsfeldsymptome bei Hirntumoren, Ophthalmologica **120**:270-285 (Nov.) 1950.

The authors present four examples of false localizing signs. In the first case, a central scotoma and concentric contraction in the field of the left eye were noted in a patient who had meningioma of the parietal portion of the falx on the left side. In the second case, upper bitemporal hemianopsia was associated with a cerebellar astrocytoma. In the third case, left homonymous hemianopsia occurred in the presence of a meningioma of the falx that had arisen at the anterior third of the sagittal sinus. In the fourth case, right homonymous hemianopsia was associated with a meningioma of the middle third of the sagittal sinus.

Binasal hemianopsia also lacks localizing value, as pointed out by Straub.[3]

Three noteworthy reports of cases of bilateral homonymous hemianopsia have appeared during the past year. Each concerns involvement of both occipital lobes. Hoff and Seitelberger [4] report on a patient with vascular hypertension who suffered from a cerebrovascular accident associated with right homonymous hemianopsia that spared the macula, right hemiparesis, alexia, and impairment of sense for numbers. The clinical diagnosis was thrombosis of the left posterior cerebral artery. Eighteen months later a second thrombus caused a left homonymous upper hemianopsia with macular involvement. Visual agnosia and alexia and disturbance of attention were present. The clinical diagnosis of fresh encephalomalacia in the region of the right posterior cerebral artery was confirmed at necropsy. A photograph and a diagram of sections of the brain show clearly the old and the recent infarcts.

Steiner [5] reports the second case, that of a woman aged 57 who had vascular hypertension and who awoke one morning unable to see on looking up, down, or to either side, and unable to get around by herself. Central acuity was normal. The preserved central isles of vision were slightly less than 5 degrees in diameter and remained unchanged for several years. Steiner ruled out a functional loss by testing at several distances and obtaining consistent results. Necropsy was not performed, but the author states the opinion that occlusion of the calcarine branch of the posterior cerebral artery was present on both sides.

Biemond [6] reports the third case, that of a woman aged 45 who had experienced hypertension for 10 years and before death had cerebral infarcts with regions of softening. She was confused and disoriented. The fields of vision were greatly restricted, but, unfortunately, the mental state of the patient did not permit accurate charting on the perimeter. Necropsy revealed regions of softening in the lateral part of both occipital lobes, which had resulted from bilateral spasm of the posterior cerebral arteries. On the right side softening extended to the occipital pole and involved only the upper part of the radiation. On the left side more extensive degeneration was present, chiefly in the middle (macular) part of the optic radiation, associated with degeneration of the macular part of the lateral geniculate body. A photomicrograph of a section through the right lateral geniculate body shows dis-

3. Straub, W.: Über die binasale Hemianopsie, Acta ophth. **30**:229-252, 1952.

4. Hoff, H., and Seitelberger, F.: Zur Klinik und Pathologie der doppelseitigen okzipitalen Herderkrankungen, Wien. klin. Wchnschr. **63**:621-623 (Sept. 7) 1951.

5. Steiner, A. A.: Homonymous Hemianopia, Bilateral with Macular Sparing, of Vascular Origin, Am. J. Ophth. **34**:1170-1173 (Aug.) 1951.

6. Biemond, A.: La projection des quadrants rétiniens dans la radiation optique, étudiée dans un cas d'hémianopsie bilatérale, Folia psychiat. neurol. et neurochir. neerl. **53**:159-164 (April) 1950.

turbance of cells in its medial part, and a section of the left lateral geniculate body shows loss of cells in the central portion. This observation confirms the earlier experimental work of Brouwer and the concept of Gordon Holmes and is a significant contribution to the anatomy of the visual pathway.

A rare complication that occurred in the course of a major surgical operation is reported by Wortham, Levin, and Wick.[7] This consisted of bilateral inferior altitudinal anopsia, which occurred during or immediately after thoracoplasty. The authors state the opinion that the loss of vision was the result of changes in the visual cortex of the superior lip of both calcarine fissures, caused by anoxia. Slight improvement in vision occurred during the 10 months that the patient was under observation.

The effects of occipital lobectomy form the subject of a comprehensive study from Paris.[8] A review of previous publications is followed by a report of five cases. The authors found that lobectomy produces homonymous hemianopsia, with a band of visual field encroaching on the half-fields that ought to be blind. The macula is included in this band, an effect attributable to overlapping of the two half-fields. Accompanying charts of the visual fields show the overlapping portions to be somewhat incongruous, an incongruity that, appearing as it does in proved cases of occipital lesions, might entitle a skeptical critic to question the accuracy of the perimetric studies on which the paper is based.

OPTIC CHIASM

Of the numerous lesions that occur in the region of the optic chiasm, meningioma may cause the greatest difficulty in early diagnosis. Joy[9] summarizes the salient features of this variety of tumor and reports a case. The patient, a woman aged 62, lost almost all sight in her left eye during a period of three or four days. Pain and tenderness on movement of the left eye appeared at the same time and persisted for several weeks. Examination three weeks after the onset disclosed pallor of the temporal portion of the optic disk. The right eye appeared normal, and its acuity and perimetric fields were normal. Roentgenograms of the skull and orbits failed to disclose any evidence of disease. Complete general and neurologic examinations revealed no abnormalities. A tentative diagnosis of retrobulbar neuritis was made. The patient was not seen again for two years. At the end of that period the left eye was completely blind and the right eye was normal. Three months later, vision of the right eye became impaired, the temporal field was contracted 15 degrees, and a small scotoma was demonstrated at the temporal side of the point of fixation. Roentgenograms failed to reveal any change in the cranial bones. After another two months visual acuity had decreased to 4/200. Angiograms disclosed elevation of the first portion of the anterior cerebral artery on the right side. A diagnosis of meningioma was made and was confirmed at operation. Part of the tumor was removed. Two months after surgical intervention, vision of the right eye was 20/20, and the field of vision was nearly normal. Vision remained normal during

7. Wortham, E.; Levin, G., and Wick, H. W.: Altitudinal Anopsia Following Thoracoplasty, A. M. A. Arch. Ophth. **47**:248-249 (Feb.) 1952.

8. Dubois-Poulsen, A.; Magis, C.; de Ajuriaguerra, J., and Hecaen, H.: Les conséquences visuelles de la lobectomie occipitale chez l'homme, Ann. ocul. **185**:305-347 (April) 1952.

9. Joy, H. H.: Suprasellar Meningioma, Tr. Am. Ophth. Soc. **49**:59-71, 1951.

the year that elapsed up to the time of the report. Joy emphasizes the slow-growing nature of meningiomas, even after they recur, and points out that angiography may be of great help in diagnosis.

In a concise report Alport [10] describes a similar problem. The patient was a woman aged 46 whose left eye had gradually become nearly blind during the preceding three years. For one year vision of the right eye had become progressively more blurred. The field of the left eye consisted of a small nasal isle; temporal contraction was demonstrated in the field of the right eye on testing with minimal stimuli. The asymmetric bitemporal hemianopsia indicated a lesion at the optic chiasm. Roentgenologic examination of the head showed no evidence of disease. On the basis of study of the visual fields alone, transfrontal craniotomy was carried out, and the neurosurgeon found a meningioma that was anterior to the chiasm and that involved both optic nerves.

From Spain [11] comes the report of an interesting case of rapidly developing blindness caused by bleeding into a tumor of the pituitary gland. The patient, a man aged 44, had a pituitary adenoma that had been treated with x-rays. He complained of sudden retrobulbar pain accompanied by bitemporal hemianopsia. Rapid progression of the visual loss occurred during the next five days, and only the nasal half of the field of the left eye was spared. At operation, an intramural hematoma was found within an eosinophilic adenoma of the pituitary. Rapid improvement in vision occurred after removal of the hematoma and adenoma. The authors state the opinion that the hemorrhage may have been the consequence of the x-ray therapy.

A report from France [12] deals with diagnostic problems as they relate to lesions of the optic chiasm. The authors suspect compression of the pregeniculate intracranial optic pathway not only in the presence of bitemporal hemianopsia but also with central scotoma, horizontal hemianopsia, or lateral hemianopsia. As diagnostic aids, they utilize roentgenography of the skull, carotid arteriography, and ventriculography. They describe the case of a man aged 27 who, after an acute infectious episode, had optic nerve atrophy and lost the upper and central portions of the visual fields. Their preoperative diagnosis was chiasmal arachnoiditis, but at operation a hypophyseal adenoma was discovered. No return of vision was noted after removal of the tumor. In another case, that of a man of similar age, bilateral central scotoma was present without peripheral contraction of the visual fields. Roentgenologic examination revealed that the sella turcica had preserved its regular contour. As in the previous case, chiasmal arachnoiditis was suspected, but an adenoma of the hypophysis was found at operation. The authors also report three cases of meningioma—one with right lateral hemianopsia, one with inferior altitudinal hemianopsia, and one with central scotoma in the left eye and, later, contraction in the superior temporal quadrant of the right eye. In these three cases are demonstrated some of the various defects in the fields that may be produced by meningiomas arising in the region of the chiasm.

10. Alport, B.: Suprasellar Meningioma: Report of a Case, Am. J. Ophth. **34**:1029-1031 (July) 1951.

11. Obrador Alcalde, S., and Urquiza Villanueva, P.: Compresión quiasmática aguda por hematoma intratumoral en adenoma de la hipófisis, Rev. españ. oto-neuro-oftal. **9**:333-337 (Sept.-Oct.) 1950.

12. Matavulj, N., and Guillaumat, L.: Quelques cas d'affections de la région chiasmatique, à périmétrie atypique, Arch. opht. **12**:67, 1952.

Chiasmal arachnoiditis continues to attract more attention in the published literature than its rare occurrence merits. Four articles concerned with it have come to attention during the past year. Dickmann, Cramer, and Kaplan [13] review much of the literature on opticochiasmatic arachnoiditis and discuss its underlying pathologic disturbances. They do not consider leptomeningitis as the lesion producing the loss of vision but state that the basic cause is a disturbance of capillary permeability resulting from reflex vasodilatation which produces exudative and cicatricial reactions. Endocrine and allergic disturbances have been found to be the principal predisposing factors. Other factors are cranial trauma, sinusitis, virus infections, syphilis, and tuberculosis. After adhesive processes have formed, surgical treatment may be of value through liberation of the optic pathways from the imprisoning leptomeningeal membranes, a procedure which may arrest the optic atrophy. On 19 of 47 patients who had operations, follow-up studies were made. Vision improved after operation in six of them. The authors stated that ideal treatment should aim at correction of the neurovascular disturbance that is the cause of the exudate, adhesions, and membranes.

Yuhl and Rand [14] report a case of tuberculous opticochiasmatic arachnoiditis in a Mexican laborer aged 25 who had incurred tuberculous meningitis. The patient improved after a long illness and extensive therapy. Nine months after the onset optic nerve atrophy was noted, together with rapidly progressing bitemporal hemianopsia. At operation, the optic chiasm and optic nerves were found to be surrounded by matted adhesions arising from the leptomeninges. These were divided and the chiasm and optic nerves liberated. Vision and visual fields returned to normal, but within six months the patient died of disseminated tuberculosis. The authors state that this case is important in that the cause of the arachnoiditis was demonstrated, whereas in many cases the etiologic agent remains obscure.

Another case in which the cause of the arachnoiditis was thought to be known is reported by Marsico.[15] His patient, a man aged 38, after an injury of the forehead, lost consciousness but next day was able to work. Four days later headaches, visual disturbances, and papilledema appeared and then soon passed away. After eight months of relative well-being, severe headaches, postneuritic optic nerve atrophy, restriction of visual fields, and diminished vision were noted. The patient refused to undergo operation. Cranial trauma is offered as the explanation.

After noting that in the majority of cases of opticochiasmatic arachnoiditis described in the literature cures are alleged to have been produced by operation, Lakatos [16] describes a case in which cure was effected by conservative management. In his case it was uncertain whether the causative agent was trauma or ethmoiditis. He states that a greatly constricted field was restored to normal by use of mercury ointment, thiamine, and parenteral injections of protein.

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13. Dickmann, G. H.; Cramer, F. K., and Kaplan, A. D.: Opto-Chiasmatic Arachnoiditis: Surgical Treatment and Results, J. Neurosurg. **8**:355-359 (July) 1951.

14. Yuhl, E. T., and Rand, C. W.: Tuberculous Opticochiasmatic Arachnoiditis: Report of a Case, J. Neurosurg. **8**:441-443 (July) 1951.

15. Marsico, V.: Su un caso di aracnoidite ottico-chiasmatica di sicura natura traumatica, Boll. ocul. **29**:729-735, 1950.

16. Lakatos, I.: Arachnoiditis optico-chiasmatica, Ophthalmologica **123**:46-50 (Jan.) 1952.

OPTIC NERVES

An extensive survey of the causes of optic neuritis has been conducted by Carroll,[17] who reports on 240 patients he has seen during the past 15 years; excluded from his report are cases of amblyopia caused by tobacco and alcohol. He classifies optic neuritis according to cause into three major groups, of approximately equal sizes: (1) optic neuritis associated with disease of adjacent structures, toxic agents, infections, Leber's disease, or vascular disease; (2) optic neuritis caused by multiple sclerosis or allied neurologic disorders, and (3) optic neuritis the cause of which is unknown. In the evaluation of therapy of optic neuritis associated with multiple sclerosis, it is important to know the course of the disease without treatment, although any controlled series is likely to be small because most patients want treatment and their physicians want to treat them. In the usual course of optic neuritis, after the onset of blurred vision, the sight frequently becomes worse for a few days, remains stationary for a period, and then begins to improve regardless of therapy. There is no specific treatment for multiple sclerosis. Carroll is not convinced that optic neuritis is benefited by use of typhoid vaccine, histamine, or antihistaminic agents. He considers it worth while to put the patient in the best possible health; for this purpose, vitamins and adequate rest are helpful. This reviewer has come to the same conclusion.

A somewhat similar survey is reported by Cordes,[18] who reviews the common and uncommon causes of optic nerve atrophy in infancy, childhood, and adolescence, dividing the cases into groups according to whether the atrophy was primary or secondary or attributable to retrobulbar neuritis. He also lists the heredofamilial diseases that produce optic nerve atrophy. In discussing each type, he cites one or two pertinent and well-chosen references. His own contribution is a survey of the etiologic factors responsible for optic nerve atrophy in 81 patients, of ages ranging from 6 months to 16 years, encountered at the University of California Hospital during a 30-year period. Of these, 43 had primary optic nerve atrophy, 32 had secondary optic nerve atrophy, and in 6 atrophy was the result of retrobulbar neuritis. The most frequent causes of optic nerve atrophy of all types were hydrocephalus (11 cases), neoplastic disease (10 cases), and congenital syphilis (9 cases). Other etiologic factors included cranial trauma, congenital optic nerve atrophy, cerebral aphasia, birth injury, congenital osseous dysplasia, osteomyelitis, hemorrhage, toxoplasmosis, spasmodic cerebral palsy, subdural hematoma, encephalomyelitis, oxycephaly and scaphocephaly, Leber's disease (hereditary optic atrophy), meningitis, and lead poisoning.

Optic neuritis with complete recovery occurred in the exanthematous phase of measles in two cases reported by Capolongo.[19] The author suggests that the pathogenesis may be related to neurotropism of the virus of measles.

Another case of visual loss in measles is reported by Whiting.[20] His patient was a 6-year-old girl who had retrobulbar neuritis in one eye only and made a complete

17. Carroll, F. D.: Optic Neuritis: A 15-Year Study, Am. J. Ophth. **35**:75-82 (Jan.) 1952.

18. Cordes, F. C.: Optic Atrophy in Infancy, Childhood, and Adolescence: A Survey of 81 Cases, Tr. Am. Ophth. Soc. **49**:365-386, 1951.

19. Capolongo, G.: Neurite ottica da morbillo, Arch. ottal. **55**:71-84 (Jan.-April) 1951.

20. Whiting, M. H.: Retrobulbar Optic Neuritis in Measles, Tr. Ophth. Soc. U. Kingdom **71**:197, 1951.

recovery. When the optic nerves are affected in measles, the involvement is usually bilateral.

An unusual case of optic neuritis is reported by Bonynge and Von Hagen [21] in a girl aged 14 who had involvement of the central nervous system during the course of an attack of infectious mononucleosis. The optic disks were edematous, and there were pain on motion of the eyes and tenderness to palpation. The fields of vision contained large central scotomas and were constricted peripherally. Treatment with dimercaprol (BAL) and aureomycin was used, and recovery was complete in three months.

Scott [22] gives a brief, but adequate, account of neuromyelitis optica, emphasizing that atypical, abortive forms occur in which the prognosis is much more favorable than reports in the literature would indicate. These favorable cases are those in which bilateral optic neuritis is present but in which signs of myelitis are minimal. He is convinced that neuromyelitis optica is an infective condition and that it is not the same as multiple sclerosis.

Two cases of bilateral transverse optic neuritis in which the course seemed to be shortened by use of cortisone are reported by Smith.[23] One patient had involvement of the spinal cord in addition to optic neuritis (neuromyelitis optica) and recovered good vision in about a month. The other patient had only optic neuritis and recovered in two weeks. The author notes that the improvement may have been coincidental but states that further trial of cortisone is warranted in similar cases. Recovery in these two cases seems to have been somewhat faster than usual. On the other hand, associates of this reviewer, after a trial of cortisone in approximately 12 cases of optic and retrobulbar neuritis, concluded that the drug had no demonstrable effect on the expected course of the disease.

Cetner [24] reports a case offering further evidence that cortisone is not effective in optic neuritis. His patient, a spinster aged 70, became blind within a few days from bilateral optic neuritis while taking 100 mg. of cortisone daily for rheumatoid arthritis. The cause of the papillitis could not be determined. The sedimentation rate of erythrocytes was increased. No evidence of temporal arteritis was noted.

In a discussion of the syndrome of compression of the intracranial portion of the optic nerve, Desvignes [25] notes that the earliest symptom is diminished vision, in the form either of central scotoma or of concentric contraction. Typically, optic atrophy is present on the affected side and papilledema on the opposite side, but bilateral papilledema may be present. The most frequent cause is tumor, particularly a meningioma arising on the lesser wing of the sphenoid bone, on the tuberculum sellae turcicae, or in the olfactory groove. Other possible causes are aneurysm of the internal carotid artery, arachnoiditis, and serous meningitis with arachnoidal

21. Bonynge, T. W., and Von Hagen, K. O.: Severe Optic Neuritis in Infectious Mononucleosis: Report of a Case, J. A. M. A. **148**:933-934 (March 15) 1952.

22. Scott, G. I.: Neuromyelitis Optica, Am. J. Ophth. **35**:755-764 (June) 1952.

23. Smith, V. L.: Bilateral Transverse Optic Neuritis: Report of Two Cases with Cortisone Therapy, A. M. A. Arch. Ophth. **46**:363-366 (Oct.) 1951.

24. Cetner, J. A.: Papillitis During Cortisone Therapy, Am. J. Ophth. **35**:713-714 (May) 1952.

25. Desvignes, P.: Le syndrome de compression du nerf optique intra-cranien, Presse méd. **59**:449-450 (April 4) 1951.

cyst. A tumor at a distance may produce compression of the optic nerves by means of dilatation of the third ventricle. It is probably logical to regard compression of the nutrient arteries of the nerve as the immediate cause of the disturbance.

A blow on the orbital rim may damage the optic nerve. Haemmerli [26] reports the case of a man aged 22 who fell against a doorpost, striking his left shoulder and the rim of the orbit. No visible signs of trauma were noted. Instantly, a black haze appeared before the left eye, and after 37 hours the left optic disk was found to be swollen and the blind spot somewhat enlarged. During the following few days small hemorrhages and some edema developed in the retina and then disappeared. Visual acuity remained good. Ultimately, the left optic disk became slightly paler than the right.

The effect of arteriosclerosis on the optic nerves is considered by several writers. Parin [27] gives an extensive review of the literature and reports two cases. He states that the ocular disturbance may be sudden or insidious and that the visual fields may show nasal or horizontal hemianopsia or concentric contraction. In acute cases, transient papilledema may be present, whereas optic nerve atrophy usually occurs in chronic cases. The pathogenesis is partly mechanical pressure by the sclerotic vessels and partly circulatory ischemia. The latter factor exerts the greater influence.

Berkson [28] reports the case of a woman aged 82 who became blind in both eyes as a result of arteriosclerotic involvement of the optic nerves. She exhibited the cupping and atrophy of the optic disks characteristic of pseudoglaucoma (cavernous optic nerve atrophy). This unfortunate outcome is rare.

The syndrome of temporal arteritis is characterized frequently by grave ocular complications—sudden loss of vision and ischemic edema of the papilla—followed later by optic atrophy. In considering this disease, Bessière and Julien [29] state that no visual improvement has been known to occur. Treatment has been unsuccessful. Amelioration of pain has been obtained by resecting the affected temporal artery. The authors report four cases, illustrate a section of a temporal artery involved in this disorder, and present an extensive bibliography.

The effect of malnutrition on the optic nerves has received an unusual amount of attention in articles published during the past year. Rodger [30] studied 238 cases of nutritional amblyopia in former Far-Eastern prisoners of war. The average time of onset of the visual loss was 11 months after capture. The onset was sudden, preceded by a few weeks of retinal teichopsia. In the initial stage the central scotoma was larger than in later stages (10 degrees, as compared with 5 degrees). Most of the late scotomas were central, absolute, and step-edged. In two cases they were annular. The only change in the fundus was pallor of the disk. The amblyopia appears to be due to endogenous toxins that damage retinal ganglionic cells or their fibers.

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26. Haemmerli, V.: Schädigung der Sehnerven nach Contusion des Orbitalrandes, Ophthalmologica **121**:69-72 (Feb.-March) 1951.

27. Parin, P.: Opticusatrophie durch Arteriosklerose der Carotis interna, Schweiz. Arch. Neurol. u. Psychiat. **67**:139-174, 1951.

28. Berkson, J.: Pseudo-Glaucoma Progressing to Blindness with Calcification of the Carotid Artery, Tr. Ophth. Soc. U. Kingdom **71**:239-242, 1951.

29. Bessière, E., and Julien, R. G.: Les manifestations papillaires dans le syndrome dit "artérite temporale." Fréquence de l'oedème papillaire ischémique, Arch. opht. **10**:701-714, 1950.

30. Rodger, F. C.: Nutritional Amblyopia: A Statistical Report of the Course and Progress in 238 Cases, A. M. A. Arch. Ophth. **47**:570-583 (May) 1952.

Obal [31] studied 39 cases of nutritional amblyopia in Germany in patients who had been prisoners of war in Russia. The general symptoms in recent cases are cachexia, diarrhea, generalized edema, polyneuritis, and hormonal disturbances. Blurring of vision occurs simultaneously in the two eyes and to an equal degree. Peripheral vision is not affected. At first there are small, negative paracentral scotomas, which coalesce within a few days or weeks to impair central vision and to form a relative central scotoma having a radius of 3 to 5 degrees for white and colored targets. Within some weeks or months, a small central isle of preserved vision, 1 or 2 degrees in diameter, appears in the middle of the scotoma. The surrounding scotomatous region condenses to a dense ring-shaped scotoma. Vision within the central isle may return to ability to distinguish small letters. Under adequate nutritional conditions, the ring scotoma breaks up into several small relative scotomas, which persist indefinitely. Disturbances of sensation of color that accompany the disease are greatest for green and yellow and less for red, while sensation for blue usually is not impaired. Adaptation to dark is seldom affected. In cases of long duration temporal pallor of the disk and granulation of the macular area are usually present.

In another article, Obal [32] points out that transverse furrowing of the fingernails appears in severe malnutrition. This sign may be used to differentiate the central scotomas of nutritional amblyopia from scotomas due to other causes, such as infections, toxins, or brain tumors.

In a study of 24 cases of amblyopia caused by tobacco or alcohol or both, Brognoli and Citterio [33] found that in many there was evidence of hepatic insufficiency, which probably was caused by the same substance as that responsible for the disease of the optic nerve. The authors give the opinion that lessened detoxicating power of the liver may play a role in the mechanism of toxic amblyopia.

The visual loss in the tobacco amblyopia of pipe smokers may be caused by swallowed nicotine, which slowly upsets gastric function and leads to metabolic derangement, according to Leishman.[34] In cigarette smoking rapid absorption of nicotine into the blood stream occurs, accompanied by inhibition of diuresis. Pipe smoking does not produce this antidiuretic effect.

Manschot and Hampe [35] discuss the origin of hemorrhage into the optic nerves in spontaneous subarachnoid hemorrhages. Of 161 patients who had spontaneous subarachnoid hemorrhage, retinal hemorrhages were observed in 34 and papilledema in 25. This incidence is much smaller than that found in other publications on the subject. The retinal hemorrhages are caused by ruptures in retinal veins from venous congestion resulting from sudden closure of the central vein where it traverses the intervaginal spaces, and are intensified by overfilling of the central

31. Obal, A.: Nutritional Amblyopia, Am. J. Ophth. **34**:857-865 (June) 1951.

32. Obal, A.: Transverse Furrows of the Nails as a Differential Diagnostic Mark in Central Scotomas, Am. J. Ophth. **35**:83-88 (Jan.) 1952.

33. Brognoli, C., and Citterio, M.: La funzionalità epatica nell'ambliopia alcoolico-tabagica, Boll. ocul. **30**:12-38, 1951.

34. Leishman, Robert,: Gastric Function in Tobacco Amblyopia, Tr. Ophth. Soc. U. Kingdom **71**:319-326, 1951.

35. Manschot, W. A., and Hampe, J. F.: Origin of Ocular Symptoms in Spontaneous Subarachnoid Haemorrhages, Acta XVI Concilium Ophthalmologicum (Britannia), pp. 356-366, 1950.

retinal artery. Blood from the intracranial subarachnoid space is pressed through an open communication in the optic canal into the subarachnoid space of the optic nerve. The authors demonstrated the existence of an open communication by means of serial sections in three cases of spontaneous subarachnoid hemorrhage and also by means of suboccipital injection of gelatin mixed with India ink into the subarachnoid space of cadavers. The ink could be traced in serial sections through the entire length of the optic nerves. The subarachnoid space in the optic canal is largest on the lateral side of the nerve; on the medial and lower sides it is distinctly smaller, and at the upper side the nerve is fixed to the dura.

In a letter, Manschot [36] points out how the findings and conclusions just noted differ from those of Walsh and Hedges,[37] published in an earlier article.

PAPILLEDEMA

The incidence of papilledema in 115 cases of cerebral tumor in Lübeck, Germany, is reported by Böke.[38] Papilledema was present in 100% of cases of tumors of the fourth ventricle, in 70% of cases of tumors of the cerebellum and cerebellopontine angle, in 60% of cases of tumors of the parieto-occipital region and brain stem, and in 25% of cases of hypophyseal tumors. These figures are somewhat greater than those of recent studies reported in the United States, where facilities for early diagnosis may be more readily available.

Tumors of the spinal cord occasionally may be responsible for choking of the optic disks. Three such cases of tumor at the level of the lower thoracic and upper lumbar vertebrae are reported by Love, Wagener, and Woltman.[39] In two cases removal of the tumor was followed by improvement of visual acuity and relief of headache. In the third case, in which the tumor of the spinal cord was too extensive for removal, cerebellar exploration revealed leptomeningitis, and symptomatic relief followed exploratory operation on the cerebellum and x-ray therapy. The authors state that the rapid recession of choked disks in these cases suggests that the internal hydrocephalus and resultant papilledema were secondary to block of the circulation of the cerebrospinal fluid at the site of the tumor in the spinal cord. They point out that in cases such as these it always will be necessary to exclude the possible presence of a separate intracranial lesion.

Five instances of papilledema were encountered among 106 cases of poliomyelitis, according to Weiman, McDowell, and Plum.[40] The papilledema developed from 11 to 48 days after the onset of the illness and persisted for an average of 60 days. The pressure of the cerebrospinal fluid was increased, and headache was present. Visual fields and visual acuity were normal. The authors suggest that the papilledema may be part of a generalized edema of the central nervous system.

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36. Manschot, W. A.: Optic Nerve Sheath Hemorrhage, Am. J. Ophth. **34**:1335-1336 (Sept.) 1951.

37. Walsh, F. B., and Hedges, T. R., Jr.: Optic Nerve Sheath Hemorrhage, Am. J. Ophth. **34**:509-527 (April) 1951.

38. Böke, W.: Untersuchungen über die praktische Verwertbarkeit okulärer Symptome bei Hirntumoren, Klin. Monatsbl. Augenh. **118**:113-133, 1951.

39. Love, J. G.; Wagener, H. P., and Woltman, H. W.: Tumors of the Spinal Cord Associated with Choking of the Optic Disks, A. M. A. Arch. Neurol. & Psychiat. **66**:171-177 (Aug.) 1951.

40. Weiman, C. G.; McDowell, F. H., and Plum, F.: Papilledema in Poliomyelitis, A. M. A. Arch. Neurol. & Psychiat. **66**:722-727 (Dec.) 1951.

Papilledema occurred after an unusual surgical procedure in the case of a boy aged 13 reported from France.[41] After diving into a swimming pool, the lad experienced progressive right hemiplegia, motor aphasia, and right homonymous hemianopsia. A diagnosis was made of thrombosis of the left common carotid artery. This condition was treated surgically by means of anastomoses between the central portion of the left common carotid artery and the peripheral portion of the internal jugular vein and between the peripheral portion of the common carotid artery and the central portion of the internal jugular vein. The hemiplegia subsided; the aphasia decreased, and the hemianopsia disappeared. Three weeks afterward the left optic disk was swollen 3 D. and the veins of the left retina appeared to contain arterial blood. On observation again, three months later, both disks were swollen 3 D.; this condition persisted, with little change. Also present were bilateral exophthalmos and congestion of the vessels of the conjunctivas, both conditions being greater on the left side than on the right.

Wagener,[42] in a review article, discusses the use of the terms "optic neuritis," "papilledema," and "choked disk" and stresses the importance of adequate estimations of vision and visual fields in all patients who have edema of the optic disks. Only in this way can the correct diagnosis be attained. He reports instances of edema of the optic disks in three cases of encephalitis, the causes of which were, respectively, nonspecific encephalitis, lymphocytic choriomeningitis, and measles. In the first and third cases optic neuritis appeared to be the best diagnosis, and in the second the diagnosis was questionable. He states that it is logical to assume that any edema of the optic disk seen in a patient who has had encephalitis should be considered as optic neuritis until proved otherwise.

PUPILS

Lowenstein,[43] in an account of some of his own methods of examination for the early diagnosis of multiple sclerosis, notes that according to traditional teaching pupillary signs are rare in this disease. His personal pupillographic studies, however, indicate that such signs are almost always present in cases of advanced disease and frequently appear early in the course. He presents four cases of early multiple sclerosis in which pupillary changes were evident in the form of difference in size of the two pupils and in the promptness of their reactions. He states that the disturbance in pupillary reaction gives the clinician an independent lesion that may be of aid in the diagnosis of this disease.

A case of Adie's syndrome is reported by Yourish.[44] The patient presented the classic signs, and the author records that instillation of 2.5% methacholine chloride

41. Petit-Dutaillis, D., and Guillaumat, L.: Évolution ophthalmoscopique après anastomose carotido-jugulaire pour thrombose de la carotide chez un adolescent, Bull. et mém. Soc. franç. opht. **63**:50-56, 1950.

42. Wagener, H. P.: Edema of the Optic Disks in Cases of Encephalitis, Am. J. M. Sc. **223**:205-215 (Feb.) 1952.

43. Lowenstein, O.: Methods for the Early Diagnosis of Multiple Sclerosis: Observations with Special Reference to the So-Called Ocular Type, A. M. A. Arch. Ophth. **46**:513-526 (Nov.) 1951.

44. Yourish, N.: Adie's Syndrome, U. S. Armed Forces M. J. **2**:1515-1517 (Oct.) 1951.

(mecholyl chloride®) resulted in prompt contraction of the tonic pupil and in no contraction of the normal pupil. This test, first proposed by Scheie, is of great help in the confirmation of a clinical diagnosis of myotonic pupillary reaction.

Two other cases of this syndrome are reported by Barré, Redslob, and Nordmann.[45] Their first case is of interest because the pupillary disturbance came on within the short period of 10 days. In their second case the pupillary disturbance apparently followed cranial injury.

Rhythmic changes in pupillary motility are described by Werner [46] in the case of a woman aged 74. For five days before she died, of uremia, cardiac hypertrophy, nephrosclerosis, and cerebral arteriosclerosis, she had been given a few small doses of morphine and barbiturates, during which time she had been unconscious. The pupils were contracted during apnea; when respiration began again, the pupils dilated rapidly, became widest in midrespiration, and then decreased in size as the rate of breathing diminished. It was considered that the mydriasis was caused by anoxemia and the miosis by a lowered state of consciousness.

Isolated internal ophthalmoplegia is reported by Holmberg [47] in two cases of herpes zoster ophthalmicus. He considers that the process was localized peripherally to the parasympathetic fibers of the sphincter pupillae.

OCULAR MOVEMENTS

Two reports of congenital bilateral paralysis of the horizontal ocular muscles and of the face (Moebius' syndrome) have appeared during the past year. Stansbury [48] reports a case in a boy aged 6 years who had complete functional loss of the sixth and seventh cranial nerves and partial involvement of the third, fourth, and fifth cranial nerves. He suggests that the cause is an agenesis or aplasia of certain motor nuclei in the brain stem but that the nature of the underlying process is unknown.

The other case, reported by Marner,[49] is that of a boy aged 10 years who had congenital bilateral paralysis in the region supplied by the abducens and facial nerves, with total failure of lateral ocular movements, limited vertical mobility of the eyes, and atrophy of muscles of the tongue. His conception of the process was the same as that mentioned by Stansbury.

Lesions of the cavernous sinus cause a characteristic syndrome, consisting of unilateral involvement of the third, fourth, fifth, and sixth cranial nerves. Huber [50] reports on 22 cases of this syndrome. Aneurysm of the internal carotid artery was the cause in 4 of these cases, thrombosis of the cavernous sinus in 2, and tumor in 16.

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45. Barré, J. A.; Redslob, E., and Nordmann, J.: Remarques sur deux cas récents de pupillotonie avec aréflexie tendineuse (Syndrome de Weill-Reys, *alias* d'Adie), Rev. neurol. **83**:138-140 (Aug.) 1950.

46. Werner, I.: Cheyne-Stokes Respiration Associated with Rhythmic Pupillary Changes: A Case Report, Acta psychiat. et neurol. **26**:213-217, 1951.

47. Holmberg, Å.: Two Cases of Isolated Ophthalmoplegia Interna in Herpes Zoster Ophthalmicus, Acta ophth. **29**:483-486, 1951.

48. Stansbury, J. R.: Moebius's Syndrome; Congenital Oculofacial Paralysis: A Case Report, Am. J. Ophth. **35**:256-261 (Feb.) 1952.

49. Marner, E.: Congenital Bilateral Sixth and Seventh Cranial Nerve Palsies Associated with Extremital Aplasia (Möbius' Syndrome), Acta ophth. **29**:129-137, 1951.

50. Huber, A.: Über das Syndrom des Sinus cavernosus, Ophthalmologica **121**:118-125 (Feb.-March) 1951.

The tumors in seven cases of the last group were metastatic carcinomas. When this syndrome is produced by aneurysm, it begins with constant pain or numbness in the region supplied by the fifth nerve, and paresis of the ocular muscles soon develops. The homolateral optic nerve may become involved later, and erosion of bone usually is evident on roentgenography. The diagnosis may be confirmed by an arteriogram.

A rare case of bilateral aneurysm in the cavernous sinus is reported by Alpers and associates.[51] This occurred in a woman aged 45 who two or three years previously had experienced noise in her head, the symptoms persisting until six weeks before her present trouble. Five weeks before entrance into the hospital she experienced severe headache and complete ophthalmoplegia on the left side. Five days before admission headache and ophthalmoplegia appeared on the right side. Examination revealed bilateral exophthalmos and ophthalmoplegia. A bruit was audible over the right side of the head. Arteriographic studies disclosed a fistula between the carotid artery and the cavernous sinus on the right side and a saccular aneurysm on the left. No surgical procedure was carried out. Movement returned spontaneously to the right eye, but ophthalmoplegia persisted on the left.

Paralysis of divergence was associated with increased intracranial pressure in five cases studied by Chamlin and Davidoff,[52] who were able to find reports of 10 similar cases in the literature. In 8 of the total of 15 cases, a focal lesion was present in the posterior fossa. In nine cases the paralysis of divergence disappeared after relief of the increased intracranial pressure. In the authors' opinion, their observations argue against the existence of a true center for divergence. Perhaps they are correct, but I am not convinced, and I cling stubbornly to my own opinion, likewise based on clinical evidence, that there is such a center.

Paralysis of divergence was also present in a case of polioencephalitis haemorrhagica, of Wernicke, in a man aged 25, reported by Cogan.[53] The patient was severely cachectic because of an intestinal disorder, for which ileotransversostomy was done; multiple vitamin deficiencies had been present for several months. The pupils reacted to light but not to near stimulus. The patient improved after treatment with large amounts of vitamins, and five years later the only ocular anomaly was esophoria of a few degrees for distance.

A study of the ocular symptoms in four cases of disease in the region of the corpora quadrigemina is reported by Schultz-Zehden.[54] The ages ranged from 7 to 18 years, and choking of the optic disks was present in all. In three cases paresis of upward gaze was noted; in two cases disturbance in the pupils was observed, and in two cases defects in the field of vision were present. The lesions in the four cases were pinealoma, tuberculoma of the pineal region, fibroglioma originating in the roof of the aqueduct, and glioblastoma arising in the pineal region.

51. Alpers, B. J.; Schlezinger, N. S., and Tassman, I. M.: Bilateral Internal Carotid Aneurysm Involving Cavernous Sinus: Right Carotid Artery-Cavernous Sinus Fistula and Left Saccular Aneurysm, A. M. A. Arch. Ophth. **46**:403-407 (Oct.) 1951.

52. Chamlin, M., and Davidoff, L. M.: Divergence Paralysis with Increased Intracranial Pressure: Further Observations, A. M. A. Arch. Ophth. **46**:145-147 (Aug.) 1951.

53. Cogan, D. G.: Divergence Paralysis and Paralysis of the Near Reflex Associated with a Malnutrition Syndrome (Presumably Wernicke's Polioencephalitis), A. M. A. Arch. Ophth. **46**:436-437 (Oct.) 1951.

54. Schultz-Zehden, W.: Augenveränderungen bei Tumoren der Vierhügelgegend, Ztschr. ges. inn. Med. **6**:8-12 (Jan.) 1951.

Eareckson and Miller [55] report a case of paralysis of the third cranial nerve with sparing of the pupil in a man aged 62 who had had diabetes mellitus and relatively mild diabetic retinopathy for seven years. The patient had had pain in the left eye for four days prior to the onset of the paresis. The fourth and sixth nerves were spared, and the condition cleared in about two months. Approximately three months after the onset of the paresis in the left eye, the right eye became involved in a similar fashion, and it, too, cleared spontaneously in about two months. Intracranial aneurysm at the circle of Willis was suspected at the time of the involvement of the first eye but was ruled out by an arteriogram. The lesions were probably hemorrhages into the nuclei of the oculomotor nerves.

In rare instances variable ophthalmoplegia may be caused by angioneurotic edema, as exemplified by the case of a woman aged 25 reported by Fry and Schlezinger.[56] The condition occurred irregularly and unpredictably and was bilateral. Edema and ptosis of the lids were prominent signs, along with limitation of ocular rotation. The authors note that this disorder is rare and that differential diagnosis of variable ophthalmoplegia must include myasthenia gravis, parasellar intracranial aneurysm, thyropituitary endocrinopathy, and cyclic paralysis of the oculomotor nerve.

Two unusual cases of myasthenia gravis are reported by Minton,[57] unusual because of the ages of the patients concerned, one patient being an 11-year-old girl, the other a 73-year-old man.

The ocular symptoms of myasthenia gravis are described by Fontaine,[58] who presents a review of 17 cases of his own. In his experience, the ocular muscles are always involved at one time or another, and in 50% of cases this is the mode of onset. The symptoms are, in order of frequency, ptosis, involvement of the orbicularis muscles of the lids, and involvement of the oculomotor muscles. The muscular weakness may be manifest only as a heterophoria. Fontaine states that accommodative difficulty deserves a place in the symptomatology of myasthenia, although he did not find the motility of the pupil to be impaired. The major characteristic of the symptoms is variability. They occur suddenly and last for indeterminate periods, while their intensity is related to the fatigue of the patient. The test with neostigmine is not certain when the disease is of long duration but is reliable when the disease is of recent origin. The irreversibility of the ophthalmoplegia after it has remained fixed for some time suggests that prolonged inaction produces structural changes.

Kiloh and Nevin [59] report five cases of progressive muscular dystrophy with early and predominant affection of the external ocular muscles. They suggest that the term "ocular myopathy" be applied to these and similar cases. In the literature, similar conditions have been termed "nuclear ophthalmoplegia," but the authors doubt whether the evidence justifies this concept.

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55. Eareckson, V. O., and Miller, J. M.: Third-Nerve Palsy with Sparing of Pupil in Diabetes Mellitus: A Subsequent Identical Lesion of the Opposite Eye, A. M. A. Arch. Ophth. **47**:607-610 (May) 1952.

56. Fry, W. E., and Schlezinger, N. S.: Variable Ophthalmoplegia Due to Angioneurotic Edema, A. M. A. Arch. Ophth. **46**:655-660 (Dec.) 1951.

57. Minton, J.: Ocular Manifestations of Myasthenia Gravis, Tr. Ophth. Soc. U. Kingdom **71**:235-238, 1951.

58. Fontaine, M.: Les symptômes oculaires de la myasthénie, Arch. opht. **12**:157-169, 1952.

59. Kiloh, L. G., and Nevin, S.: Progressive Dystrophy of the External Ocular Muscles (Ocular Myopathy), Brain **74**:115-143 (June) 1951.

Hooper [60] reports on the orbital complications that occurred in 58 of 500 cases of cranial injury. In 10 cases visual impairment caused by loss of accommodation was noted. Defects occurred in the visual fields in 27 cases; in 14 of these the initial loss of vision in one eye was complete. In three cases the loss took place after the first examination following injury. In one of these three cases it occurred within the first 24 hours; in the second case it appeared three days after injury, and in the third the eye became blind gradually during the six weeks following injury. Partial defects of the visual fields were extremely varied. Paralyses of extrinsic ocular muscles occurred in 19 cases, as follows, multiple paralyses being present in some: oculomotor nerve, 12; trochlear nerve, 2, and abducens nerve, 12. In all but one case in which the oculomotor nerve alone was injured impact occurred in the frontal region. In all but one case in which the abducens nerve alone was injured the impact was from the side. The oculomotor nerve is injured usually in the orbit or the sphenoidal (superior orbital) fissure. The abducens may be involved in this location or at the apex of the petrous pyramid. Diplopia may also be caused by displacement of the globe resulting from damage to the bony orbit.

SYNDROMES

The phacomatoses include tuberous sclerosis (Bourneville's disease), neurofibromatosis (Recklinghausen's disease), angiomatosis of the retina and cerebellum (Lindau-von Hippel disease), and nevus flammeus (Sturge-Weber syndrome). These are described in a compact, yet comprehensive, review by Kirby.[61] He lists 50 pertinent references and discusses the similarities and dissimilarities that exist among the various lesions in these disorders.

A detailed account of angiomatosis of the retina and cerebellum with a short historical review is presented by Saebø.[62] He reports on a family in which five members in two generations suffered from the disease. Both eyes were affected in all cases. Therapeutic results of cautery to the retinal lesions were poor. The author describes the typical changes in the fundus as consisting of pairs of vessels, artery and vein, leading to angiomatous tumors. The vessels are large and irregular in caliber, and the color of the vein tends to approach that of the artery. Small white patches, resembling those of circinate retinitis, are seen. Stress is laid on the importance of examination of the peripheral portions of the retina in all neurologic cases, for, although Saebø does not say so, he correctly implies that in early stages small angiomas may be visible only at the far periphery, when the larger vessels still appear normal. The ease with which these lesions are overlooked may account for the high proportion of monocular involvement recorded in the literature. All the five patients described by Saebø had changes in the retina: in addition, three had cerebellar tumors, and one had a cyst of the epididymis.

Møller [63] presents a pedigree in which five verified cases of Lindau-von Hippel disease appeared in two generations, with four additional cases suggestive of the disease scattered over three generations. The inheritance appears to be that of an

60. Hooper, R. S.: Orbital Complications of Head Injury, Brit. J. Surg. **39**:126-138 (Sept.) 1951.

61. Kirby, T. J.: Ocular Phakomatoses, Am. J. M. Sc. **222**:227-239 (Aug.) 1951.

62. Saebø, J. A.: Von Hippel-Lindau's Disease, Acta ophth. **30**:129-153, 1952.

63. Møller, P. M.: Another Family with von Hippel-Lindau Disease, Acta. ophth. **30**:155-165, 1952.

irregular dominant. He emphasizes the variable clinical picture in this disorder and states that in some families angiomas form in the retina or cerebellum and in others they occur as cysts of the pancreas or kidney.

A single case of this disorder is reported from France,[64] that of a man aged 30 who had experienced headaches, nystagmus, and disturbed equilibrium. The optic disks were choked, but the retinal vessels appeared normal. A cystic angioblastoma was excised from the cerebellum. Three years later polycystic disease of the kidneys was found, and a year after that, when the patient complained of diminution of vision in the right eye, signs of retinal angiomatosis were noted. The left eye was normal. As pointed out by the authors, the presence of cerebellar tumor or visceral cysts demands that the fundus be examined minutely for possible early retinal angiomas.

Djacos and Joannidès[65] present three cases of the Sturge-Weber syndrome. After reviewing many theories, they note that the pathogenesis of the disease remains obscure. In 80% of the cases, roentgenologic examination of the cranium discloses a characteristic picture, with shadows localized in the superficial cortex of the brain, especially in the occipital lobes, in the form of tortuous or parallel lines. On the basis of their gonioscopic studies, they conclude that the glaucoma in the Sturge-Weber syndrome must be due to excessive production of aqueous at the level of the choroidal angioma, because the angle of filtration is free and the head of pressure of the flow of aqueous is normal, proving the filtration system to be intact.

MISCELLANEOUS CONDITIONS

A noteworthy review of the ocular manifestations of multiple sclerosis is presented by Wybar,[66] who encountered retrobulbar neuritis in 25 of 91 cases. In 19 of the 25 cases it was the first symptom of multiple sclerosis, preceding other symptoms by an average of 8 years; the longest interval was 20 years. About half the patients recovered good vision (6/12 or better). In eight of the cases retrobulbar neuritis was bilateral. No optic nerve was affected more than once, and the author states that he knows of no case of recurrence being recorded. This is a startling statement. Because Wagener[67] is at present reviewing a series of cases of retrobulbar neuritis associated with multiple sclerosis, he was requested to comment on Wybar's statement. He finds evidence of acute recurrences in 5 of 39 cases of retrobulbar neuritis associated with multiple sclerosis. Of these, the recurrences in three were mild. In only two cases was the loss of vision intense, and in neither was the patient examined during the episodes of blindness, the evidence being based on the history as given by the patient. In nine other cases mild recrudescences occurred during the course of chronic retrobulbar neuritis, but these could scarcely be called "acute recurrences." In light of this experience, it would appear that a recurrent episode of acute retrobulbar neuritis in a previously involved eye actually is a rare event, and Wybar's experience is not unique after all.

64. François, J., and Coffyn, J.: Angiomatose retino-cerebello-viscérale de von Hippel-Lindau, Ann. ocul. **184**:206-213 (March) 1951.

65. Djacos, C., and Joannidès, T.: La maladie de Sturge-Weber-Krabbe, Ann. ocul. **184**:994-1014 (Nov.) 1951.

66. Wybar, K. C.: Ocular Manifestations of Disseminated Sclerosis, Proc. Roy. Soc. Med. **45**:315-319 (May) 1952.

67. Wagener, H. P.: Personal communication to the author.

Diplopia occurred in 28 of Wybar's series of 91 cases of multiple sclerosis. In 14 of these it was the first evidence of disease, preceding other manifestations by an average of four years. Nystagmus was present in 57 cases. In half of these the nystagmus was horizontal, occurring either alone or with vertical or rotary components. In most of the other half there was "ataxic" nystagmus, by which Wybar means internuclear ophthalmoplegia. He found this associated often with vertical nystagmus. The optic disks disclosed pallor in 23 cases. Localized sheathing of some of the retinal veins was present in nine cases, in all but one of which it was bilateral. This finding is especially gratifying to me, because it is the first published confirmation that retinal venous sheathing does occur in about 10% of cases of multiple sclerosis. To be found, it must be diligently sought through dilated pupils and in a darkened room.

Wybar made a special study of the pupil in his entire series of 91 cases. Anisocoria was present in 11, but in 7 of these the difference in size was only 0.5 mm. In 19 cases slight sluggishness of the reflex to light was noted. For accurate study of the pupillary reactions, he used a method proposed by Stern in 1944, in which oscillations of the pupil are induced by a narrow beam of light. Abnormal pupillary reflexes were found by this method in half the entire series of patients with multiple sclerosis. In some the defect lay in the afferent conducting pathways, and in others it was in the complex efferent pathways. The significance of these minimal pupillary disturbances is yet to be evaluated.

A splendid discussion of the syndrome of crocodile tears is presented by Chorobski.[68] Even his footnote on the origin of the term is fascinating. He includes a comprehensive review of the literature and several diagrams to indicate the possible pathways involved. He presents one case, that of a patient who had facial paresis complicated by profuse lacrimation, or crocodile tears. Tearing appeared only when the patient took hot soup, and upset emotions with weeping caused tearing only on the nonparetic side. The condition disappeared spontaneously after a few months. As an explanation, the author suggests interaction of the adjacent autonomic and taste fibers of the facial nerve because of inadequate insulation during the period of recovery.

It has generally been believed that the cornea has the property of receiving the sensation of pain, but not that of touch, although the evidence is not unequivocal. Cogan and Ginsberg,[69] in order to locate the representation of corneoconjunctival sensation in the central nervous system, studied a man who had a clear-cut syndrome of thrombosis of the posterior inferior cerebellar artery. The sudden onset had been one week prior to the first observations. Findings were typical and included right-sided cerebellar ataxia, dysphonia, nystagmus, right-sided Horner syndrome, persistent hiccup, absence of sensations of pain and temperature with preservation of sensation of touch on the right side of the face, and anesthesia of the left limbs and the left side of the trunk. Examination of the eyes revealed that the cornea and bulbar conjunctiva of the right eye were anesthetic to all test objects, including those

68. Chorobski, J.: Syndrome of Crocodile Tears, A. M. A. Arch. Neurol. & Psychiat. **65**:299-318 (March) 1951.

69. Cogan, D. G., and Ginsberg, J.: Representation of Corneal and Conjunctival Sensation in the Central Nervous System, A. M. A. Arch. Ophth. **47**:273-275 (March) 1952.

for pain, temperature, and touch stimulation. From this it is inferred that sensation in the cornea and bulbar conjunctiva is represented in the brain stem with sensation of pain and temperature in the face.

A study of photophobia is reported by Lebensohn.[70] He concludes that photophobia may accompany a lesion in any region supplied by the ophthalmic division of the trigeminal nerve. It is manifest most frequently in disease of the anterior part of the eye but may accompany cranial injury, subarachnoid hemorrhage, meningitis, acromegaly, trigeminal neuralgia, or migraine. It is induced by constriction of the iris to light. The pupillary reflex to light becomes painful after the iris has been sensitized by trigeminal axonal reflexes that produce vasodilatation and hyperalgesia. This antidromic vasodilatation is dependent on the integrity of fibers of the trigeminal nerve and vasodilator fibers whose cells of origin are in the Gasserian ganglion. It is alleviated by instillation of epinephrine, phenylephrine hydrochloride (neo-synephrine® hydrochloride), mydriatics, and inhalations of trichloroethylene.

70. Lebensohn, J. E.: Photophobia: Mechanism and Implications, Am. J. Ophth. **34**:1294-1300 (Sept.) 1951.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

George F. J. Kelly, M.D., *Chairman*

M. Luther Kauffman, M.D., *Clerk*

March 20, 1952

EXOPHTHALMOS ASSOCIATED WITH DISTURBANCES IN THE PITUITARY-THYROID AXIS

Experimental Considerations. Dr. William McK. Jefferies, Cleveland.

A review of the problems involved in the study of exophthalmos in experimental animals, with a résumé of the findings derived from this type of investigation up to the present time, was presented. After a brief discussion of the various agents which had been used to produce exophthalmos in animals and the reasons that guinea pigs have proved to be the most suitable subjects for this type of experimentation, the evolution of an accurate instrument to measure intercorneal distance in animals and a reliable technique for producing exophthalmos in guinea pigs were described. Studies of the mechanism of production of exophthalmos by injections of pituitary extract were then reviewed. Several investigators have found that an increase in the content of fluid, fat, and connective tissue of the retro-ocular tissues is involved and that the fraction of pituitary extract containing thyrotropic hormone shows the greatest exophthalmic activity. Nevertheless, by the use of pituitary extract in which the thyrotropic factor has been almost, if not completely, inactivated by iodination, it has been shown that exophthalmic effects in guinea pigs cannot be attributed to activity of the thyrotropic hormone alone. Further studies of a similar nature have indicated that the production of exophthalmos is not directly related to the fat-mobilizing effects of pituitary extract. Other experiments have exonerated corticotropin (adrenocorticotropic hormone) and follicle-stimulating hormone of any direct relationship. Hence, although the exophthalmic factor exists in close association with thyrotropic hormone in pituitary extract, it is not this hormone, and its true nature remains obscure.

The Clinical Problem. Dr. Edward Rose.

The author described briefly the ocular changes of Graves's disease, with mention of their variability and the gradations of their severity. Lack of knowledge concerning the pathogenesis of these changes is indicated by the numerous theories which have been advanced, the more important of which he reviewed. He analyzed his clinical experience with a number of patients followed for long periods and from this experience drew certain inferences regarding the classification of the syndrome. This experience casts doubt on the acceptability of the separation of exophthalmos into thyrotropic and thyrotoxic types. The frequency of allergy in patients with this syndrome invokes the possibility of local tissue hypersensitivity of the orbital contents as a factor in its pathogenesis. He reviewed endocrine and other nonsurgical methods of treatment and emphasized the difficulty of evaluating such therapeutic measures.

The Neurosurgical Problems. Dr. Rudolph Jaeger.

It is of practical interest that the neurosurgeon does not see cases of exophthalmos in association with gross surgical disease of the hypophyseal area. Adenomas of the pituitary gland, destructive disease of this body from cancer or abscess,

tumors of Rathke's duct, and tumors of the hypothalamic region do not cause proptosis. Only invading meningiomas or malignant tumors of the retro-orbital bone cause protrusion of the eyeball by their local congestion and mass increase of the periorbital and intraorbital structures.

The neurosurgeon is concerned with (1) preservation of vision through the prevention of corneal ulcerations and opacities and (2) improvement in the patient's appearance. The first may be effected by suturing the eyelids together over almost the full length of the lids after denuding the lid edges, care being taken not to disturb the puncta lacrimalia. Removal of the sutures after 10 days will leave the lids tightly grown together and assures the optimal condition for corneal healing. Since spontaneous recovery is often possible, this maneuver may preserve vision until time effects the cure, when the condition is otherwise inoperable.

The second neurosurgical maneuver for the treatment of this deformity is intracranial decompression of the orbit by removal of its roof (Naffziger operation). In severe cases this can easily be combined with removal of the lateral orbital wall (Krönlein operation), so that the maximum decompression possible is assured. When the proptosis is severe and shows no tendency to subside, this operation should be performed. It carries only a slight risk, and the cosmetic result is good. The operation may be performed on the two sides at the same time.

Slides were presented showing (1) the technique of closure of the eyelids, and (2) a patient with malignant exophthalmos before and six and nine months after closing of the lids. Cure was spontaneous. The patient was elderly, and had high blood pressure and diabetes. The technique of intracranial decompression, with the appearance of the patient before and after operation, was shown by motion pictures.

The Ophthalmological Problem. Dr. Edmund B. Spaeth.

From the standpoint of surgical therapeutics, it is necessary to differentiate the basic etiologic factors, that is, to consider whether there is a thyrotoxic or a thyrotropic factor, or whether possibly both factors are present. Ocular signs and symptoms of the thyrotropic state can quite properly be called pituitary exophthalmos. In cases with the characteristics of both a thyrotoxic and a thyrotropic state, the thyroid dysfunction, if that is present, fails to depress pituitary secretion; as Herman said, "Obviously, these cases will present the most therapeutic problems." These three states have different surgical indications, which are not related wholly to degrees of severity in the basic disease. They are essentially different diseases.

The fundamental pathologic process in the orbit for the production of exophthalmos is of significance. In thyrotropic exophthalmos the involvement of the eye, in its progression, is entirely dissociated from a thyrotoxicosis. By reason of the disturbed balance of the hormones, faults in water storage occur. Because of the confined nature of the orbital space, this may have particularly disastrous consequences in that the exophthalmos may progress inexorably. Thyrotropic exophthalmos is associated with discomfort. Pain, discomfort, lacrimation, photophobia, and diplopia frequently occur. Patients with this type of exophthalmos often consult an ophthalmologist first. He may find periocular edema, chemosis, and injection of the conjunctivas. Absence of lid spasm permits free eversion of the lids, and the degree of proptosis is more real than apparent. Often, a thick ridge of edematous tissue projects, like a finger, toward the inner canthus. Actually, in thyrotropic exophthalmos, the eyes, not the basic disease, seem to present the major therapeutic problem.

Thyrotropic exophthalmos is always a serious condition—it is not only difficult to diagnose, but equally difficult to treat. To illustrate, in a recent patient not only is the disease now quiescent, but he is in a lamentable condition. The right eye was enucleated for absolute glaucoma due to massive x-ray therapy, given because of a

faulty diagnosis of retrobulbar neoplasm; the left eye is now immobile, as a result of the muscle degeneration, and presents secondary glaucoma, though stabilized with miotics, and the lids are united in permanent intermarginal tarsorrhaphy.

Before the consideration of local surgical measures, that is, surgery with the sole purpose of saving the eye, other therapeutic procedures available must be briefly discussed. These are, first, the administration of thyroid to decrease the output of thyrotropic hormone by the pituitary, and, second, the modification of the metabolism of the hormone-producing cells of the pituitary, as by x-ray therapy, in an attempt to decrease the output of this hormone. Neither is the ophthalmologist's problem, but both are procedures to be used alone or as a part of the total treatment. For example, simple tarsorrhaphy will prevent damage to the cornea in a severely proptosed eye, regardless of the type of exophthalmos, but neither tarsorrhaphy nor orbital decompression may be sufficient to save the globe in a case of steadily developing intraorbital pressure. The exophthalmos may become so severe, even before the globe is endangered from corneal perforation, as to cause marked permanent degeneration of the orbital muscles, swelling of the optic nerve, and retinal detachment. Long-standing edema may destroy ultimately all orbital muscle tissue, terminating in degeneration and in ocular immobility. A common finding in these cases, frequently confusing as to diagnosis, is the monocular nature of the exophthalmos early in the course of the disease. This finding may result in a faulty diagnosis of unilateral retrobulbar neoplasm or pseudotumor.

The surgical technique for correction of the oculomotor paralyses is well understood. Resections are to be performed upon the paretic muscles. The sequence of surgical procedures is a bit more exacting. The basic muscle degeneration characteristic of this condition makes operation upon the conjugate yoke muscles in the form of recessions unwise, in fact, rather ineffective.

The stationary exophthalmos of arrested thyrotoxicosis, or of severe stationary, and nonmalignant, thyrotropic exophthalmos, is perhaps largely cosmetic in its demands. It may, however, be otherwise. The progressive type of malignant exophthalmos has an essential surgical aspect. It may be not only eye-saving, but even life-saving. Such a condition is frequently a tragic affair and demands both surgical and medical therapy, which frequently is heroic in extent.

The surgical treatment of lagophthalmos, and this is frequently applicable also to stationary exophthalmos requiring surgery for cosmetic reasons, is the classic levator recession of Goldstein. This operation attacks the condition on the same anatomical grounds as those used for correction of paralytic ptosis. It is ideal in quiescent cases. The levator is detached from the tarsal plate and recessed posteroward as it lies above the superior rectus muscle, and below the septum orbitale, permitting a lengthening of that portion of the lid anterior to the new detachment of the levator to the orbicularis and fascia. One can easily achieve a recession of any amount up to 10 mm. The operation may be performed through the skin surface or through the conjunctival surface, as one wishes. It seems that the operation, when done through the conjunctival surface, has a bit less postoperative reaction.

In some cases stationary exophthalmos, when unaccompanied by lid spasm, can be treated by a surgical procedure which is somewhat easier and which has a much shorter period of necessary hospitalization. This is the use of the buried silk suture of Axenfeld. It is a simple procedure and should correct a moderate degree of involvement. Braided white silk is to be used, and one must be meticulous as to surgical asepsis. I have had failures with this operation in several instances because of a stitch abscess. Such a complication will nullify the entire operation and make necessary removal of the suture.

Mention must be made also of the angle tarsorrhaphy of Wheeler. It is especially valuable in that one can adjust the surgery necessary for corrections of the upper and lower lids to dissimilar degrees, depending upon which of the two needs more correction.

The surgical correction for the thyrotropic form of exophthalmos is essentially that of orbital decompression. Cases are not uncommon in which a combination of the various surgical procedures is to be seriously considered. By this is meant the simultaneous use of a levator recession, for instance, and an angle tarsorrhaphy, or a levator recession and an orbital decompression, or the decompression of the orbit combined with angle tarsorrhaphy. Occasion has been found to utilize all three of these possibilities.

Much has been written about the transfrontal decompression. This is not for my consideration. The subzygomatic decompression, as outlined by Shugrue, is a satisfactory procedure. The orbitotomy of Krönlein is not necessary. In the Shugrue technique, one must be certain not to enter the cranial cavity, through the sphenoidal bone or to injure the maxillary nerve at the lower part of the operative field. The purpose of an orbital decompression, as Herman stated, "is to relieve edema and congestion in the orbit, and thereby to restore adequate drainage of the extraocular tissues as well."

Malignant exophthalmos, as such, can be satisfactorily treated with the Shugrue orbital decompression. No treatment other than orbital decompression is successful in this serious situation. In my experience, there is no great difference in the results obtained with the transfrontal approach and the uncapping of the roof of the orbit as it lies in the frontal fossa, and those with the subzygomatic approach and orbital decompression, as just described.

Cases have been presented in which a transfrontal-orbital decompression has been done on one orbit and a subzygomatic decompression on the other. There seemed to be no difference in the degree of correction obtained.

In summary, the surgical procedures which have to do with the ocular manifestation of thyroid dysfunction are essentially four: (1) a levator recession; (2) an angle tarsorrhaphy; (3) treatment of the muscle disturbance, with the usual sequence of necessary surgical procedures for this condition, and (4) orbital decompression, when indicated.

DISCUSSION

Dr. George F. J. Kelly: It is not quite clear to me just what Dr. Jaeger has done. From the picture, it is evident that the skull was opened on the right side and that the right orbital roof was partially removed. The picture further showed improvement in the position of both eyes. I should like to know whether both orbits can be reached from the one side, or whether this patient had two separate operations.

Dr. Rudolph Jaeger: He had a second operation. One may do the operation on the two sides at the same time, but this man had two operations.

Dr. Francis Heed Adler: This is one of the clearest expositions of an extremely difficult subject that I have heard or read; but, perhaps because of its clarity and honesty, it takes away one of our secure diagnostic props. The work of Mulvaney made us confident that we had a means by which we could differentiate so-called thyrotropic exophthalmos and thyrotoxic exophthalmos, and thereby aid the clinician in deciding in what cases it was admissible to do thyroidectomy and in what cases it was probably wise to withhold surgical treatment.

Dr. Rose has pointed out that there is not a sharp line of distinction between these two types of exophthalmos, and that the basal metabolic rate may be elevated in both—in fact, generally is. We have been led to suppose that the cases of thyrotropic exophthalmos were characterized by edema of the lids and conjunctiva,

rapidly increasing exophthalmos, and frequently associated paralysis of the ocular muscles. In unilateral cases the paralysis of the affected eye generally concerns the muscles of upward gaze. As ophthalmologists, we have a particular problem in having to separate these cases of unilateral, so-called thyrotropic exophthalmos from cases of orbital tumor, and also from cases of so-called chronic orbital myositis, described by Dunnington, Berke, and others. I have always suspected that the last-mentioned disease is, in reality, due to thyrotropism. To abolish these two clean-cut entities, even though cases are seen which fall between the two extremes, will make it more difficult for the ophthalmologist to give an opinion as to the cases in which the condition will progress if thyroidectomy is done. Nevertheless, we have to face facts, and I hope that before long those who have been working on this problem will have something better to substitute than Mulvaney's scheme, which it now appears does not have too firm a footing.

DR. IRVING H. LEOPOLD: The orbital tonometry of Copper has been suggested as helpful in the differential diagnosis of these conditions. Have any of the panel of discussers had personal experience with this diagnostic tool?

Is there a difference in incidence of malignant exophthalmos after the use of propylthiouracil, radioactive iodine, or thyroidectomy for control of signs of an overactive thyroid?

DR. ALFRED COWAN: It is known that persons with exophthalmos often go blind with glaucoma. Of course, the glaucoma is secondary. In spite of this, we never hesitate to use cycloplegics in cases of proptosis or exophthalmos. Perhaps some one can explain why glaucoma is not produced in these cases.

DR. WILLIAM MCK. JEFFERIES: I appreciate the referral of these clinical questions to me, since my experience with exophthalmos has not been confined entirely to guinea pigs.

My colleagues and I also have encountered unpredictable effects in these patients. I have seen three treated with radioactive iodine. One was a man aged approximately 40, with proptosis, chemosis, ophthalmoplegia, and moderate hyperthyroidism, who had a basal metabolic rate of + 40%—a classic picture of "malignant exophthalmos." After receiving radioactive iodine therapy, he failed to return to the clinic for about six months, at which time he had the classic features of myxedema; yet his eyes were greatly improved. Hence, the fact that the occurrence of myxedema in these patients is not invariably detrimental to their ocular status suggests that the behavior of the thyrotropic hormone is not the whole story. Nevertheless, a sufficiently large number of patients with ophthalmopathic Graves's disease have experienced aggravation of their exophthalmos upon becoming hypothyroid to indicate that it is best to avoid this development. The other two patients who received radioactive iodine therapy in our clinic experienced improvement in their ophthalmopathy, but I have heard of one patient so treated elsewhere whose eyes subsequently became worse.

Subtotal thyroidectomy seems to be the therapy most likely to be followed by worsening of the eyes, so that it is generally considered to be contraindicated in cases of exophthalmos. The administration of antithyroid drugs, such as propylthiouracil, which increase stimulation, theoretically would not be good for the eyes, and there have been reports of aggravation of ophthalmopathy while patients were receiving them. Hence, we agree with Dr. Rose in preferring to use a combination of potassium iodide and thyroid in treating patients with Graves's disease.

The reports that practically every patient experiences an increase in prominence of the eyes after subtotal thyroidectomy might be explained by a concomitant gain in body weight. Unfortunately, these changes in eye measurements in human subjects have not been correlated with the body weight. It is possible that the slight increase in prominence of the eyes under such circumstances may be a manifesta-

tion of better general nutrition rather than a result of increased pituitary stimulation. In none of the patients so followed in these reports did severe ophthalmopathy actually develop.

We tried cortisone in a dose of 100 mg. daily for two weeks in one patient with ophthalmopathic Graves's disease, without appreciable effect. Other workers have encountered similar disappointments with corticotropin and cortisone after the initial encouraging reports. These two hormones, therefore, do not appear to be significant additions to therapy in this puzzling clinical picture.

DR. EDMUND B. SPAETH: To answer Dr. Cowan's question relative to "the reason for the onset of secondary glaucoma, in these conditions" I should like to defer to Dr. Mann in her analysis of ocular pathology in exophthalmos. I think her explanation is reasonable. She likened it to the secondary glaucoma from orbital conditions associated with venous conditions, the fibroses, pulsating exophthalmos, and pseudotumor. This is not the secondary glaucoma resulting from a perforation of the globe. In such a situation anything may occur. Dr. Mann's thought was that the glaucoma was due to closure—slow, inexorable and progressive—of the major venous channels in the orbit, as represented by the vortex veins. This is the only answer I have.

Obituaries

RICHARD GORDON SCOBEE, M.D.
1914-1952

Richard Gordon Scobee died of coronary occlusion at his home in St. Louis on Sunday evening, June 22, 1952, at the age of 37.

He was born in Sherman, Texas, on Nov. 14, 1914, and soon moved to El Paso, where he spent most of his boyhood years. Later he moved with his parents to Houston, Texas, and attended Rice Institute, receiving an A.B. degree from that school in 1935. He then entered the University of Texas School of Medicine at Galveston and graduated with the degree of Doctor of Medicine in 1939. A year's rotating internship was served at the Methodist Hospital in Fort Worth, Texas, before he came to St. Louis in July, 1940, for his specialized training in ophthalmology. Finishing his resident training in ophthalmology at Washington University School of Medicine and Barnes Hospital in July, 1942, he was appointed to the full-time teaching staff as assistant in ophthalmology at Washington University. In September, 1943, he was granted a leave of absence to enter the Air Force of the United States Army. He was assigned to the Department of Ophthalmology of the School of Aviation Medicine at Randolph Field, Texas, where he served until he was placed on inactive duty in September, 1945. Returning to St. Louis, he was appointed an instructor in ophthalmology and the director of graduate training in ophthalmology at Washington University School of Medicine. He was advanced to the rank of assistant professor in 1948.

Dr. Scobee's interest in ophthalmology began before his medical-school days, for he served as office assistant to Dr. E. W. Griffey, of Houston, Texas, during the summer months of 1935, 1936, and 1937. His enthusiasm for ophthalmology did not lessen during his medical-school years, but during his internship our specialty almost lost one of its keenest students, for he became intensely interested in obstetrics. If his "first love" had not been so enduring, we might not have had today a textbook on "The Oculorotary Muscles," but perhaps a volume on some phase of obstetrics.

The voids in the field of ocular motility were first confronted by Dr. Scobee during his tour of duty at Randolph Field during World War II. He was immediately bombarded with questions concerning normal and abnormal muscle balance for which there were no adequate answers available—only the opinions of those who in the past had been asked similar questions. His problem was to supply the answers, and this he did most admirably. His opportunity for basic clinical research in this field was a golden one, and he took full advantage of it. There were adequate clinical material, plenty of clerical help, and statistical advice, so that the data which he obtained could be presented in their most useful form. The many reports published by the Air Force on his work attest his unusual drive and energy.

Problems in motility continued to be his primary interest after he returned from active duty with the Air Force in 1945. As a part of the graduate course in ophthalmology at Washington University, he prepared a series of lectures on motility,

which, after revision, appeared in late 1947 as a textbook on "The Oculorotary Muscles," the second, revised, edition appearing only shortly before his death. His work on problems in motility continued when he took over the "muscle clinic" at the university, and, with financial support from the Navy, many excellent papers on heterophoria and heterotropia appeared in the journals. In the field of squint he soon became a champion of the anatomic, or mechanical, factor in the etiology of the condition; though not denying the presence of innervational factors, he

RICHARD GORDON SCOBEE, M.D.
1914—1952

believed that too many were overlooking the mechanical effects of anatomic anomalies of the muscle insertions. He described and named several of these anomalies and constantly looked for them in his surgical work.

In his work with heterotropia, he quickly became aware of his neglect of the sensory aspects of the deviation, and in 1948 he was able to procure the assistance of a trained orthoptist. With an increasing volume of work, additional help was obtained, and in 1949 students were accepted for training in orthoptics. His enthusiasm for orthoptics and his past record led to his appointment to the American

Orthoptic Council in 1948. In 1950 he was instrumental in organizing and establishing instruction courses for members of the American Association of Orthoptic Technicians, which are held during the Academy meeting each October. In 1951 he furnished the inspiration for the *American Orthoptic Journal,* assisted in the arrangements for its publication, and became its first editor. He was also in charge of the summer didactic course for orthoptic technicians given by the American Orthoptic Council the past two summers, but his illness and death prevented active participation in either course.

These words recite but a few of Dick Scobee's activities, for he was a man with many irons in the fire. His drive and energy were unbounded. He was a popular and forceful speaker and participated in the scientific programs of many organizations. His accomplishments in thirty-seven years of life were more and greater than most of us will have attained in life's full span. His impact on ophthalmology, his associates, his students, and his friends was profound and will not soon be forgotten. To lose Dick Scobee was a tragic event in the lives of us all, especially for those of us who knew him best.

He was married to Mary Frances Guthrie in January, 1941, and is survived by his wife, his two children, and his parents.

DAVID M. FREEMAN, M.D.

News and Comment

EDITED BY W. H. BENEDICT

GENERAL NEWS

Louis Bothman Fellowship in Ocular Pathology.—Establishment of the Louis Bothman Fellowship in Ocular Pathology at the University of Illinois College of Medicine has been announced by Dean Stanley W. Olson.

The university has received a grant in the amount of $5,000 from the Louis Bothman Memorial Fund, Inc., of Phi Delta Epsilon fraternity in support of the fellowship in ophthalmology.

The late Dr. Bothman held the rank of clinical professor of ophthalmology at the University of Illinois. He was instrumental in developing a laboratory in ocular pathology at the University's Research and Educational Hospitals.

Annual Postgraduate Course in Ophthalmology, State University of New York at Syracuse, Medical Center.—The Department of Ophthalmology of the State University of New York at Syracuse Medical Center will present its third annual postgraduate course in ophthalmology at the Hotel Syracuse, Friday and Saturday, Dec. 5 and 6.

The following guest lecturers will participate: Albert D. Ruedemann, M.D.; F. Bruce Fralick, M.D.; Harold G. Scheie, M.D., and Raynold N. Berke, M.D.

The tuition fee will be $25, payable to the State University of New York at Syracuse Medical Center, Irving Ave., Syracuse, N. Y. This fee covers tuition, daily luncheons, and dinner Friday night. The course will be limited to 60 members. These will be accepted in the order in which applications, accompanied by checks, are received. Inquiries regarding the course may be addressed to Harold H. Joy, M.D., 504 State Tower Building, Syracuse, N. Y.

Books

Le champ visuelle. By A. du Bois-Poulsen. Masson et Cie, 120 Boulevard Saint-Germain, Paris, 1952.

This volume is an authoritative treatise on perimetry. The author covers all phases of perimetry in 1,175 pages, and for those who read French it will be a very valuable reference book.

Ultraviolet Radiation. By Lewis R Koller. Price, $6.50. John Wiley & Sons, Inc., 440 4th Ave., New York 16, 1952.

The author of this monograph on ultraviolet radiation is a research associate in the General Electric Research Laboratories at Schenectady, N. Y. The book is written for the basic scientist and the industrialist who find it necessary to work in this portion of the spectrum.

Of special interest to the ophthalmologist is Chapter VII, which deals with some applications and effects of ultraviolet radiation. This chapter has sections on the production of erythema, conjunctivitis, ultraviolet radiation in the prevention and cure of rickets, production of vitamin B, germicidal effects, effects on viruses and other organisms, and the disinfection of air, water, and other liquids. Fluorescent lamps are also discussed.

Ocular Surgery. By H. Arruga. Translated by Michael J. Hogan and Luis E. Chaparro. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 18, 1952.

The Spanish edition of this book was one of the most valuable treatises we have had on the surgery of the eye and its adnexa, owing to the magnificent illustrations. The translation into English has made it that much more valuable to English-speaking ophthalmologists who do not have a reading knowledge of Spanish.

The translators are to be congratulated on the excellence of the style. Most translations can be recognized by the too long and involved sentences. This criticism does not apply to this book, which is written in fluent, readable English. No more beautiful illustrations have ever appeared in an English text.

Progress in Ophthalmology and Otolaryngology. By Meyer Weiner and Edward Maumenee (Part I, representing Ophthalmology) and Percy E. Ireland and Joseph A. Sullivan (Part II, representing Otolaryngology). Grune & Stratton, Inc., 381 4th Ave., New York 16, 1952.

This volume, which is issued as a quadrennial review, carries on the excellent service afforded by "Ophthalmology in the War Years," which covered the literature from January, 1940, to June, 1946. There are 72 contributors, all of whom are well known in their respective fields.

Progress in ophthalmology is dealt with in four separate headings: (1) The Basic Sciences in Ophthalmology; (2) Diagnosis and Treatment of Diseases of the Eye; (3) Surgery of the Eye; (4) Related Subjects in Ophthalmology.

In each of these main divisions various phases of ophthalmology are brought up to date. All the manuscripts in this issue are uniformly of a high caliber. The authors are to be congratulated on the general excellence of this volume.

Geometry and the Imagination. By D. Hilbert and S. Cohn-Vossen. Translation by P. Nemenyi. Chelsea Publishing Company, 231 W. 29th St., New York 1, 1952.

This is a translation into English of the famous "Anschauliche Geometrie," by Hilbert and Cohn-Vossen. The basis of the book is a course of lectures which were given at Göttingen in 1920, and the purpose of the lectures was to give a presentation of geometry in its visual intuitive aspects. The book will appeal to ophthalmologists who have a basic interest in geometry. It has no practical applications for the clinician.

Principles and Methods of Physical Diagnosis. By Simon S. Leopold, M.D. Price, $7.50. Pp. 430, 11 in color with 390 illustrations, 19 color plates. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1952.

This book is an excellent and concise text on the examination of the patient designed for students in their second year in the medical school. Of particular interest to ophthalmology is the section on examination of the eyes. The book will serve as a useful outline for the student's introduction to the eye clinic.

Between Two Worlds: The Memoirs of a Physician. By Benjamin L. Gordon, M.D. Price, $4. Pp. 354. Bookman Associates, Inc., 34 E. 23d St., New York 10, 1952.

Dr. Gordon, who has conducted a successful ophthalmologic practice for over forty years, first in Philadelphia and then in Atlantic City, writes the recollections of a widely varied life experience. Born in a ghetto community in Lithuania, he gives a picture of his childhood steeped in the traditions of Jewish

lore and scourged by Czarist persecution. He came to this country in the early 1890's and describes movingly the economic hardships and spiritual triumphs of his struggle for education and his early days as a general practitioner.

Training in ophthalmology he obtained at the Jefferson Hospital and in Dr. Oliver's Clinic at Wills Hospital, but he recounts little of this experience. Clearly, the dominating interest in his life was in Jewish affairs, and he tells of his participation in the early beginnings of the Zionist movement, of his visit to Palestine in 1904, and of his meetings with many Jewish leaders. The book is fluently and modestly written.

Pharmacology in Clinical Practice. By Harry Beckman, M.D. Price, $12.50. Pp. 839, with 152 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1952.

The author has made a unique approach to this subject by grouping the drugs discussed under the various specialties. Drugs used in ophthalmology occupy sixteen pages. This section is well documented and brought up to date, and will be found useful by all ophthalmologists.

Color in Business, Science, and Industry. By D. E. B. Judd. Price, $6.50. Pp. 401. John Wiley & Sons, Inc., 440 4th Ave., New York 16, 1952.

This book is divided into three parts. Part I outlines the elementary principles of the eye as a sense organ and the basic facts of color vision, including types of color blindness and methods of testing. Part II deals with tools and techniques, such as spectrophotometry, standards in colorimetry, and the reproduction of pictures in color. Part III deals with physics and the psychophysics of colorant layers. The book will be useful for all who deal in colors, particularly the manufacturer.

The book is a happy combination of science and common sense and is written by one of the outstanding scientists in this field.

The Oculorotary Muscles. Edition 2. By Richard G. Scobee, M.D. Price, $11. Pp. 512, with 159 illustrations. C. V. Mosby Company, 3207 Washington Blvd., St. Louis 2, 1952.

It is fortunate for ophthalmology that Dr. Scobee was enabled to publish this second edition of his well-known text on the oculorotary muscles before his untimely death. The first edition of this book has been widely read and accepted as one of the best American texts on muscles. This revision supplies us with those changes Dr. Scobee saw fit to make as the result of his experience with the book in several years of teaching.

In his preface Dr. Scobee states that many portions of the second edition were discussed with Dr. Walter B. Lancaster, and that "the keen reader will perceive the results." The section on Functional and Surgical Anatomy has been considerably enlarged. Various portions on physiology, such as Retinal Correspondence, the Horopter, Sherrington's and Listing's Law, and recent ideas on proprioception have been rewritten. Dr. Scobee's recent work on the heterophorias has been included, and a new section on intermittent exotropia has been added.

This volume will continue to be an extremely valuable text and will serve to keep with us for many years the genius of one who gave a great deal to ophthalmology.

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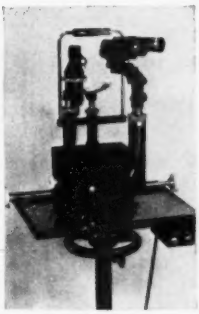

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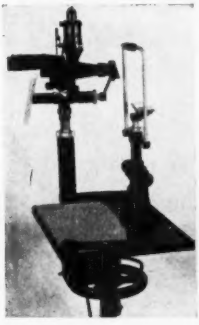

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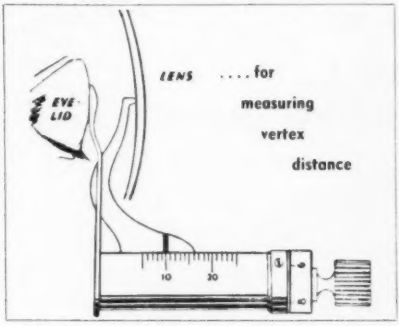

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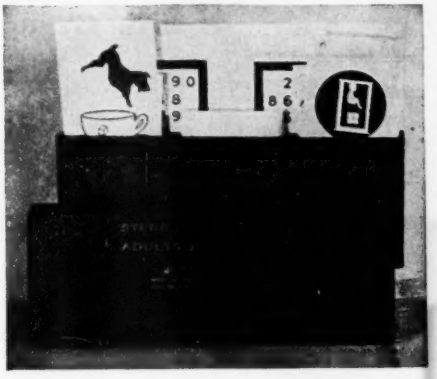

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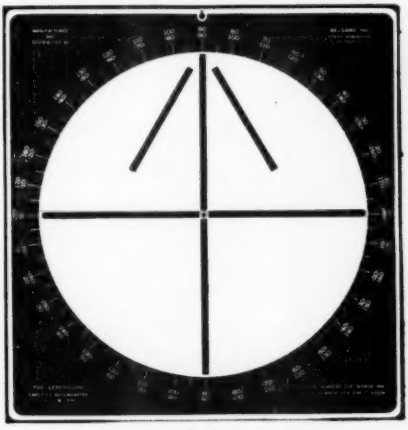

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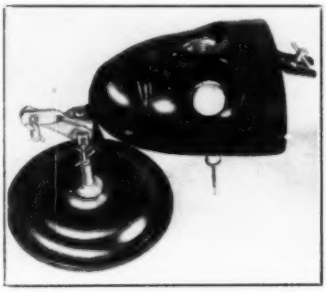

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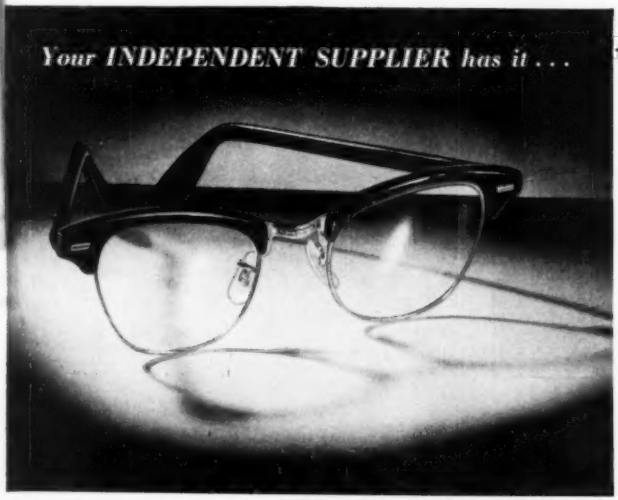

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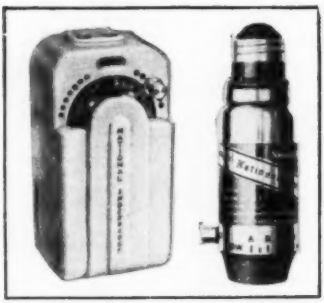

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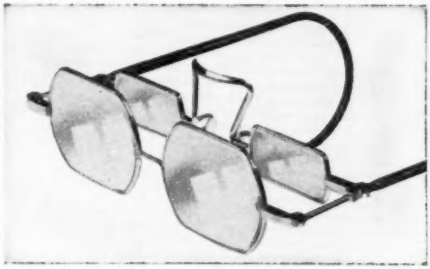

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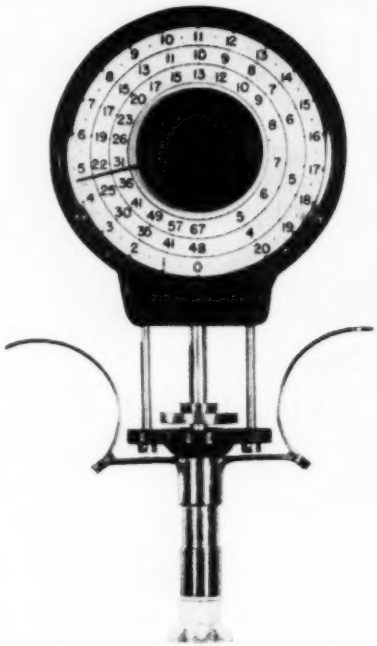

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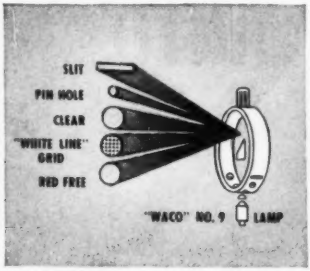

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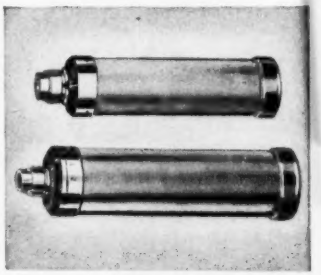

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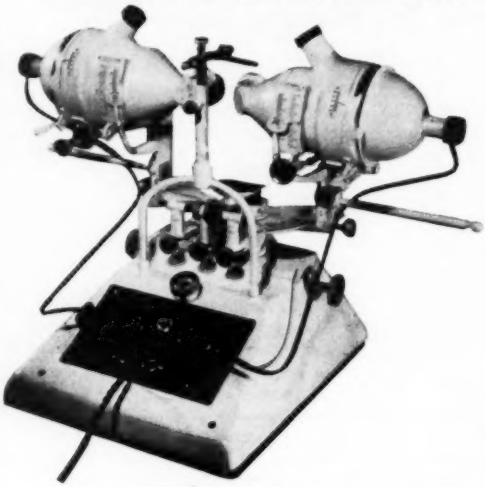

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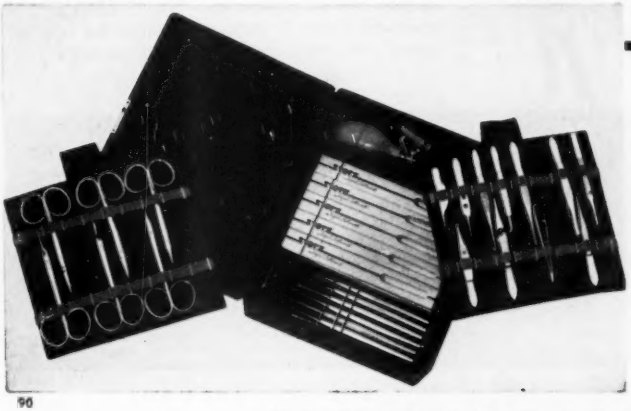

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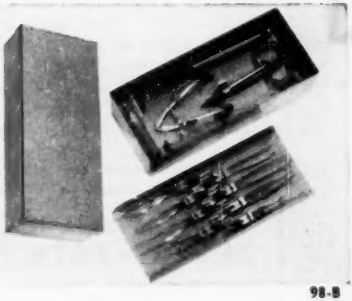

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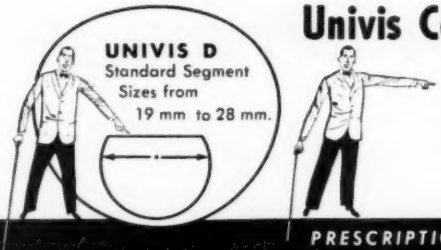

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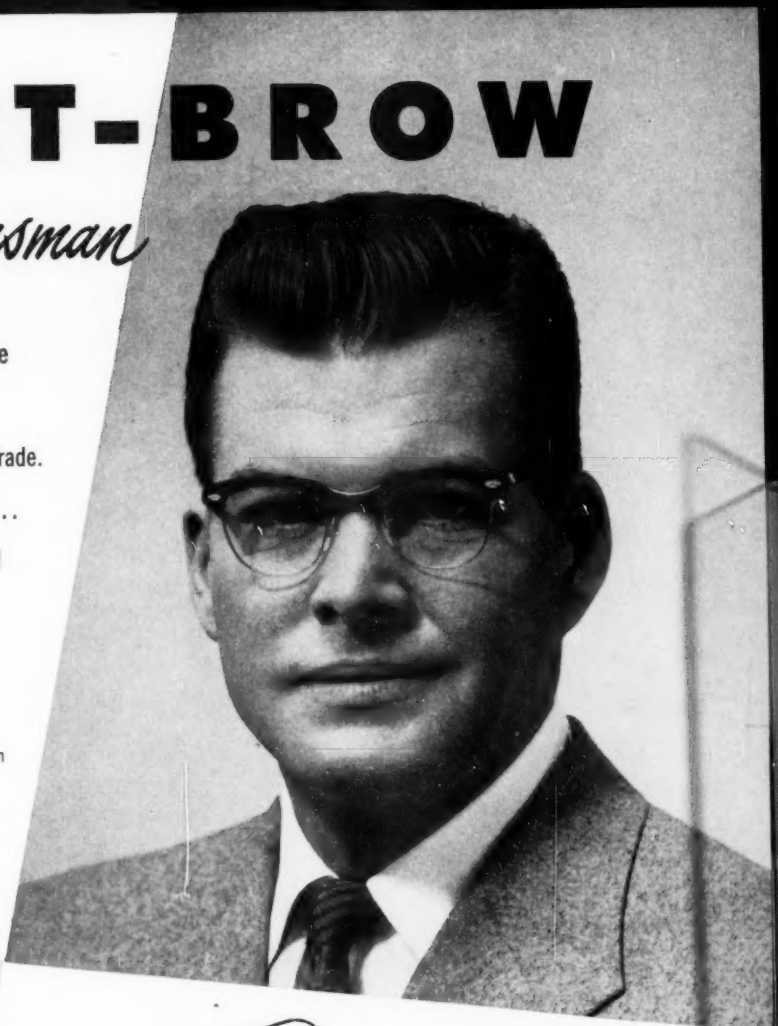

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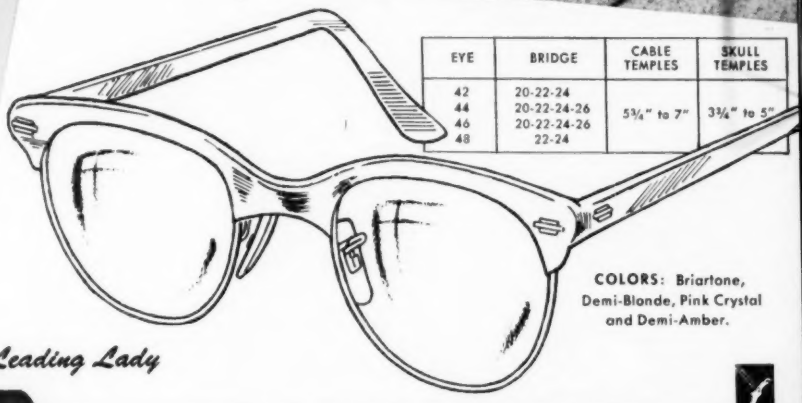

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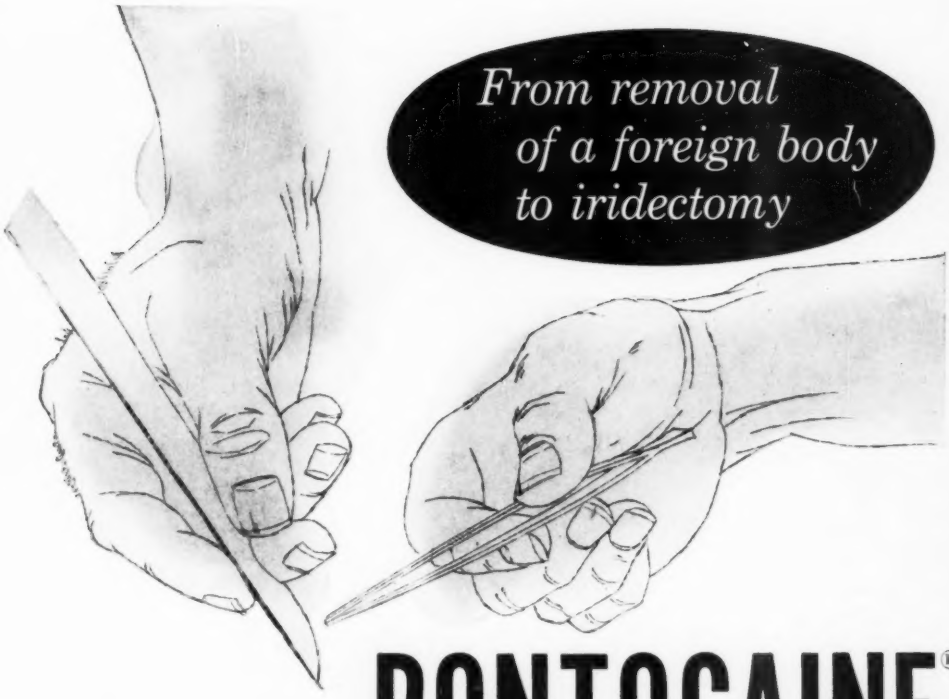

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